CORONARY ARTERY DISEASE IN FEMALES

DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT OF

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M.D. DEGREE IN GENERAL MEDICINE (BRANCH I)

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CHENNAI

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CERTIFICATE

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"Thankfulness and praise to the Almighty
For His blessings throughout this work"

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ACRONYMS

STEMI	- ST Elevation Myocardial Infarction
NSTEMI	-Non ST Elevation Myocardial Infarction
MI	- Myocardial Infarction
BMI	- Body Mass Index
CHF	-Congestive Heart Failure
CAD	-Coronary Artery Disease
Wt	-Weight in kilogram
Ht	-Height in centimeter
CP	-Chest Pain
HT	-Hypertension
DM	-Diabetes Mellitus
KS	-Killip Score
JVP	-Jugular Venous Pressure
BP	-Blood Pressure
ECG	-Electrocardiogram
AWMI	-Anterior Wall Myocardial Infarction
ASMI	-Antero Septal Myocardial Infarction
LMI	-Lateralwall Myocardial Infarction
IWMI	-Inferior Wall Myocardial Infarction
PWMI	-Posterior Wall Myocardial Infarction
RVMI	-Right Ventricular Myocardial Infarction
LDL-C	-Low Density Lipoprotein Cholesterol

HDL-C	-High Density Lipoprotein Cholesterol
TC	-Total Cholesterol
TG	-Triglycerides
CRP	-C-Reactive Protein
S1	-First Heart Sound
S2	-Second Heart Sound
S3	-Third Heart Sound
S4	-Fourth Heart Sound
ESR	-Erythrocyte Sedimentation Rate
VSD	-Ventricular Septal Defect
IV	-Intra Venous
BT	-Bleeding Time
CT	-Clotting Time
PT	-Prothrombin Time
AF	-Atrial Fibrillation
VF	-Ventricular Fibrillation
LVD	-Left Ventricular Dysfunction
SBP	-Systolic Blood Pressure
APTT	-Activated Partial Thromboplastin Time

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Abstract

Title: Coronary artery disease in females.

Objective:

1. To find the various modes of presentation and clinical profile of coronary artery disease in females
2. To study the associated risk factopors

Design: Prospective cross sectional study.

Place: Coimbatore Medical College Hospital, Coimbatore, Tertiary Care Hospital.

Period of study: September 2010 to June 2011

Inclusion criteria: 1. Patients above 40 years

2. Hypertension

3. Diabete mellitus

4. Dyslipidemia

Exclusion criteria: 1. Congenital heart disease

2. Rheumatic heart disease

3. Structural heart disease

4. Electrical abnormalities

Subjects: 100 female patients admitted with symptoms, signs and ECG changes suggestive of CAD with biochemical markers taken as cases.

Results: In this study, 33% were among age group between 60 - 70 years. Coronary artery disease mortality among women gradually increases with age and increase in the risk of coronary artery disease is related to a higher incidence of hypertension, diabetes, obesity, and dyslipidemia.

Conclusion:

1. The most common presentation is chest pain.
2. Waist-hip ratio associated with obesity and overweight increases the risk of myocardial infarction in female populations.
3. Most common presentation is STEMI.
4. Systemic hypertension, diabetes mellitus also associated with increased trisk of MI

Keywords: Coronary artery disesae, females, systemic hypertension, diabetes mellitus, obesity.

1. INTRODUCTION

Coronary heart disease has been defined as “impairment of heart function due to inadequate blood flow to the heart compared to its needs, caused by obstructive changes in the coronary circulation to the heart”.

Coronary heart disease is assuming serious dimension in developing countries. It is expected to be the single most important cause of death in India by the year 2015.

Coronary artery disease is the leading cause of death among women, regardless of race or ethnicity and causing the deaths of 1 in 3 women than from stroke, lung cancer, chronic obstructive lung disease, and breast cancer combined.

Women with coronary artery disease present differently than men, have different pathophysiologies and risks profiles and are often significantly older and thus often have poorer outcomes.

Experts in industrialized societies have long recognized that the first presentation with coronary heart disease occurs approximately 10 years later among women than men, most commonly after menopause.

Although coronary artery disease in general manifests earlier in less well-developed countries, the approximate 8 to 10 years age gap in time of onset between men and women is universal.

Despite this delay in onset, mortality from coronary heart disease is increasing more rapidly among women than men from both developed and developing world.

2. AIM OF STUDY

1. This study is to find the various modes of presentation and clinical profile of coronary artery disease in females.

2. To study the associated risk factors, there is an urgent need to recognize all these conditions so as to reduce the burden associated with it in terms of increased morbidity and mortality.

3. REVIEW OF LITERATURE

DEFINITION:

Coronary heart disease has been defined as "impairment of heart function due to inadequate blood flow to the heart compared to its needs, caused by obstructive changes in the coronary circulation to the heart".

Depending on the rate of development and ultimate severity of the arterial narrowing(S) and the myocardial response, four ischemic syndromes may results.

1. Angina pectoris, of which there are three variants, the most threatening being unstable angina.
2. Myocardial infarction, the most important form, in which there can be substantial myocardial damage.
3. Chronic ischemic heart disease with CHF.
4. Sudden cardiac death.

Many women die every year from cardiovascular disease than from any other cause, yet women worry more about breast cancer than heart disease. Women with heart disease may present differently than men, have unique underlying pathophysiologies, and have distinctive risk benefit profiles with commonly accepted therapies[1].

Heart disease is far more age dependent in women than in men; women with cardiovascular disease are older and have more co morbidities. This fact, in turn, make diagnostic and treatment procedures more problematic in women. In addition, many effective pharmacological strategies are underutilized, and there is a lack of gender-specific date on numerous therapies. The fact that

heart disease is on the decline in men but not women highlights our failure to treat this large segment of the population optimally.

The aetiology of atherothrombotic cardiovascular disease is multi-factorial, and several 'risk factors' are recognized to predispose an individual to develop the disease.

These cardiovascular risk factors, which were initially characterized in the Framingham Heart Study, include: age, family history of premature cardiovascular disease, smoking, hypertension, hyperlipidaemia, diabetes, obesity and sedentary lifestyle.

Although a family history of cardiovascular disease is a recognized risk factor, it is important to emphasize that cardiovascular disease is polygenic and numerous genetic abnormalities have been implicated in the development of the final common disease state. Furthermore, expression of disease is often closely linked to environmental risk factors such as smoking, diet and physical inactivity.

In addition, the disease does not present until middle age, or later, in most cases. Clearly, therefore, the 'burden of disease' in a population is strongly associated with the prevalence of recognized risk factors within that population. Incidence and prevalence rates for cardiovascular diseases depend to a large extent

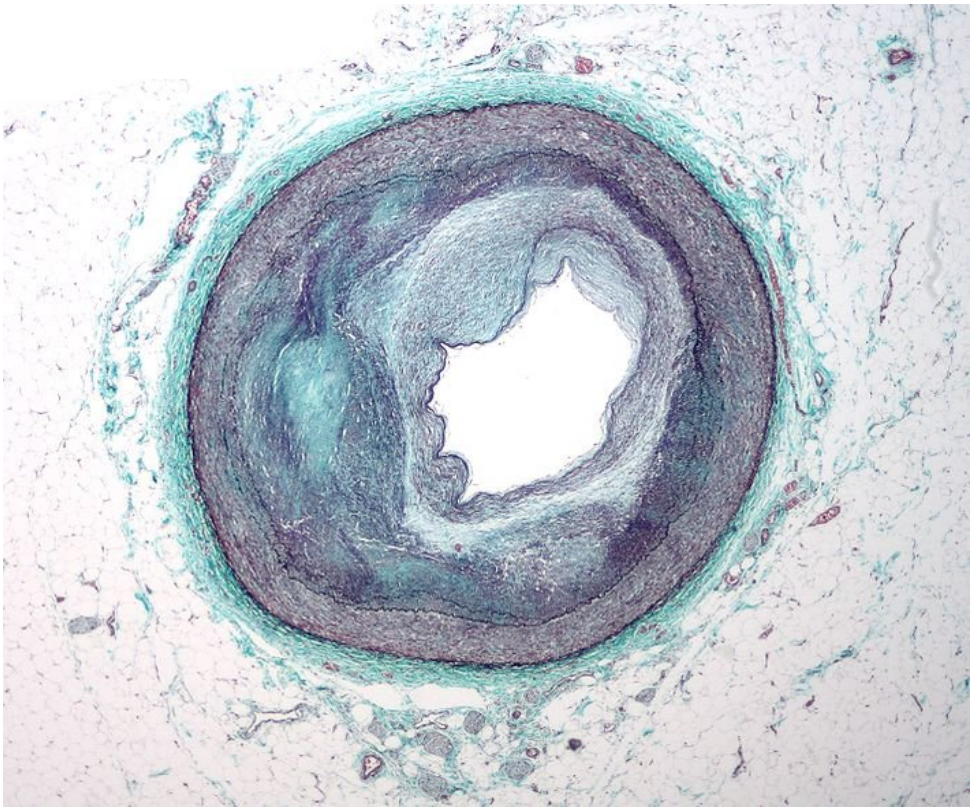


Figure 3.1 Atheromatous Plaque

on the age profile of the population, socio-economic, dietary and other lifestyle patterns; although other influences, including genetic differences influenced by ethnicity, are also important.

RISK FACTORS FOR CORONARY HEART DISEASE IN WOMEN AND THEIR MODIFICATION

Diabetes Mellitus and Metabolic Syndrome

Diabetes is associated with a greater incremental risk in women, completely eliminating the "female advantage." The American Heart Association awards double weight to diabetes in women[(2)] when calculating CHD risk, similar to the weight given a systolic blood pressure of 173 mm Hg or above or cholesterol level of 316 mg/dl or above. More than in men, diabetes

dramatically increases the mortality of myocardial infarction in women. Type 2 diabetes[3] is associated with obesity, abdominal body fat distribution, hypertension, atherogenic dyslipidemia, and insulin resistance, all of which have been associated with higher CHD risk.

This complex of abnormalities, termed "metabolic syndrome," alters hepatic metabolism, lipoprotein levels, and circulating insulin levels. More so than in men, obesity and body fat distribution appear to be independent coronary artery disease risk factor in women[4]. Diabetes is also linked with endothelial dysfunction and a variety of platelet abnormalities[5]. Data from the Diabetes Control and Complications Trail suggest that intensive diabetes therapy reduces cardiovascular complications in men and women younger than 40 years.

Hypertension

More than 25 million American women have high blood pressure, and cardiovascular risk related to hypertension[6] rises steeply with age in females. Further, although women have fewer cardiovascular events, the population risk attributable to hypertension is higher for women[7] than men because of the increased incidence with age and the longevity of women. Nonpharmacological interventions effectively decrease blood pressure in women, including a low-salt diet, physical activity, and weight loss. However, compliance with such changes is low (~10 percent) and blood pressure increases again when physical activity decreases or weight is regained.

Most trails have shown equal efficacy of blood pressure lowering to prevent cardiovascular events in men and women. The seventh Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure includes a single set of guidelines for both

men and women, stating that “large, long-term clinical trials of antihypertensive treatment have not demonstrated clinically significant gender[8] differences in blood pressure response and outcomes.” Although it is clear that hypertension in women should be treated as aggressively as in men, it is possible that the optimal choice of antihypertensive agent may differ.

The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial(ALLHAT) demonstrated superiority of diuretic therapy in the prospecified female cohort, as in the trial as a whole. The Second Australian National Blood Pressure Study Group found that angiotensin-converting enzyme (ACE) inhibitor therapy decreased cardiovascular endpoints relative to hydrochlorothiazide in men but not in women. The numbers of women included in trials are often too small to draw conclusions.

Smoking

Tobacco contributes to 17 percent of all female deaths in the United States and results in more deaths from CHD and stroke than any other cause. The combination of accelerated atherosclerosis and propensity to vascular thrombosis induced by cigarette smoking is responsible for a six-to nine-fold increased risk of myocardial infarction among female smokers compared with nonsmokers. There is a similar increase in stroke risk. The combination of cigarette smoking and oral contraceptive use appears to be particularly potent at increasing the risk of arterial thrombosis.

Cigarettes have an antiestrogenic effect and induce an unfavorable lipid profile, leading women to lose their “natural” protection against atherosclerotic vascular disease. Currently available methods to assist with quitting may be less effective in women, perhaps because of a

greater behavioral component and less nicotine addiction in women smokers. Environmental exposure to tobacco smoke increases the risk of cardiovascular disease in women, and assessment of environmental exposure is an important part of risk assessment.

Lipids

The average lipid profile women are affected by hormonal status and changes throughout life. Young women have lower low-density lipoprotein (LDL)[(9)] cholesterol levels and higher high-density lipoprotein (HDL)[(10)] cholesterol levels than men of the same age. As women age, LDL cholesterol increases, HDL cholesterol decreases, and the risk of CHD climbs. Elevated total cholesterol and LDL levels are only weakly associated with CHD in women and only in women 65 years old or younger. Instead, HDL cholesterol is closely and inversely associated with CHD[(11)] risk.

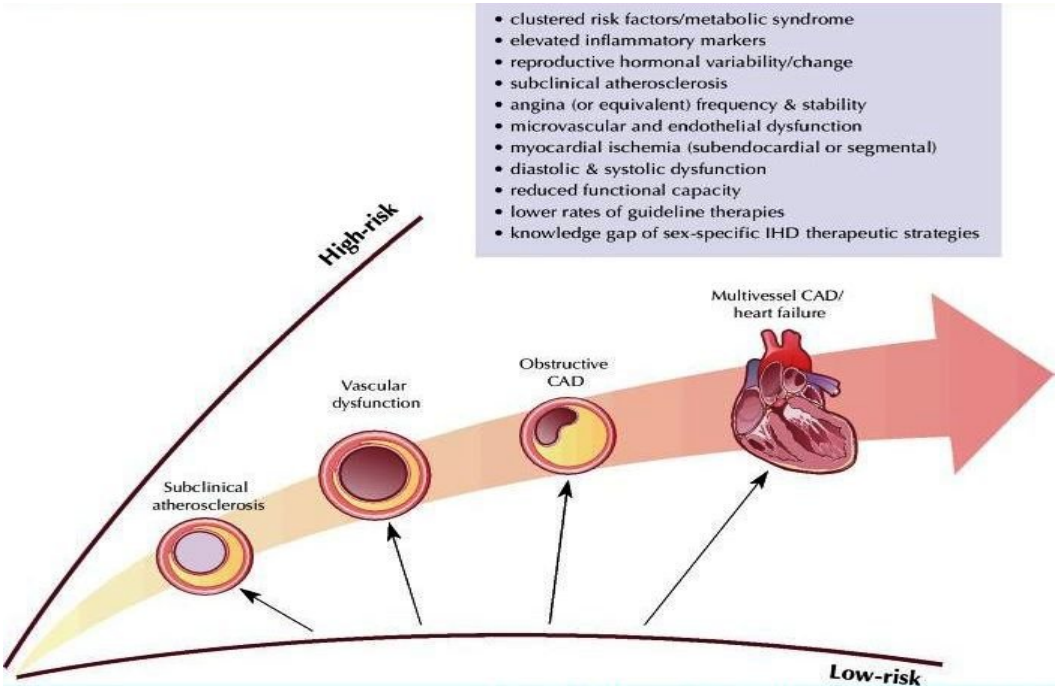


Figure 3.2 Risk in CAD

Triglycerides[12] are an independent predictor of CHD, particularly in older women. Lipoprotein(a), a composite of LDL, apolipoprotein B-100, and apolipoprotein(a), is also associated with higher cardiac risk in women. Initial modification of a high-risk lipoprotein profile is generally accomplished by the same life-style changes and medications in men and women, although dietary interventions may be less effective in women.

Multiple trials have demonstrated efficacy of 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase inhibitors[13], or statins in both primary and secondary prevention of coronary events and death in women with both elevated and normal cholesterol, supporting the persistent theory that there are benefits of these agents independent of LDL-lowering effects.

Women have generally not been included in trails of other classes of lipid-lowering agents, such as the bile acid sequestrant cholestyramine and the fibrate gemfibrozil.

In addition, there are no data from large trails on the efficacy of lipid-lowering agents targeted more specifically at altering the HDL and triglyceride lipid subfractions, which appear more important in women. It seems reasonable to apply strategies shown to be successful in men, recognizing that optimal care of women with dyslipidemia may eventually be different from that of men[14].

Estrogen

The presence of estrogen in the premenopausal female population, the obvious protection this group enjoys against cardiovascular events, and documentation of estrogen receptors in cardiomyocytes and vascular tissues in both men and women led tremendous enthusiasm for use of postmenopausal hormone replacement therapy as a preventive measure against atherosclerotic heart disease. This enthusiasm was bolstered by multiple observational studies suggesting

improved longevity and decreased cardiac events in postmenopausal women receiving hormone replacement therapy as well as mechanistic data supporting biological plausibility[15].

Multiple randomized controlled trails in the past 3 years have refuted this hypothesis and provided strong evidence of an increase in cardiovascular risk particularly in the first year after beginning therapy, combined with increased risk of breast cancer, thromboembolic disease, and stroke, resulting in a withdrawal of prior recommendations.

In the Women's Health Initiative Study, use of estrogen alone, without a progestin, neither caused nor prevented cardiac events, although it did increase the risk of stroke and decrease the risk of hip fracture. Other studies documenting progression of atherosclerosis during hormone replacement therapy have confirmed the lack of benefit. Hormone replacement therapy has no place in prevention of heart disease in women at present.

Many questions about estrogen remain unanswered. Would alternative formulations of either estrogen or progesterone be of benefit? Does starting therapy immediately at menopause eliminate risk? Can genotype predict the risk-benefit profile of estrogen therapy? In addition, if the estrogen hypothesis is indeed a fallacy, what does afford the protection in the premenopausal female population? Several candidates have been suggested, although none have substantial scientific support: lower iron levels and higher oxytocin levels, for example. Estrogen therapy is associated with impressive changes in so many risk factors, including lower LDL, higher HDL, improved glucose tolerance, and reductions in weight and waist circumference, that an understanding of the mechanism of increased risk is likely to revolutionize yet again our understanding of the atherosclerotic process.

Diet and Obesity

Excess caloric intake and obesity are a growing epidemic worldwide, and more than one-third of American women are classified as obese. The Nurses' Health Study revealed a sevenfold higher cardiovascular mortality in the heaviest women, and Framingham Offspring study demonstrated a dramatic rise in risk factors for cardiovascular disease at body mass indices above 20.

Obesity is associated with elevated-reactive protein (CRP), particularly in women. The combination of obesity and diabetes appears particularly deadly in women, as does the pattern of fat distribution. Abdominal fat accumulation is an important predictor of type 2 diabetes mellitus, hypertriglyceridemia, hypertension, and CHD[16] . Among women, a waist-to-hip ratio greater than 0.88 is predictive of a substantially increased risk of cardiovascular events, as is a waist circumference of more than 38 inches[17].

Physical Activity

Physical Activity[18] is more prevalent among women than men. There is a strong inverse association between physical activity and coronary events in women. Physical activity also has a salutary effect on other cardiovascular risk factors, including hypertension, obesity, and diabetes mellitus.

The effect of regular exercise to increase HDL cholesterol and include weight loss may be less in women than in men. Significant barriers to regular exercise exist for American women, particularly older women. Caregiving duties, low energy, and lack of peers seen exercising are cited as impeding women's compliance with exercise recommendations.

Inflammation

The close relationship between inflammation and cardiovascular risk is discussed in depth elsewhere. Baseline CRP levels predict future cardiovascular risk in healthy women, particularly those metabolic syndrome. Although we currently lack clinical trial evidence in support of CRP as a target of therapy, the ability of statins to reduce CRP is associated with benefit in lipid-lowering trials and ability of oral estrogen to raise CRP is associated with harm.

Psychosocial Factors

The interaction of psychosocial and behavioral factors and heart disease is complex and has not been rigorously studied. Several cardiovascular risk factors are related to behavior (obesity, smoking, exercise), yet modification may be more difficult for women than for men, with caregiving roles often blamed for this failure.

Perceived stress and lack of situational control have been found to increase CHD risk in both genders. Social networks and support influence CHD outcome both independently and through the likelihood of compliance with therapeutic strategies and their impact may be greater in women, who are more likely to live alone. Although depression increases cardiac risk in both women and men, a causal relationship is unclear, as is the impact of treatment.

Emerging Risk Factors

Many newer markers of increased cardiovascular risk apply equally to men and women, including abnormal endothelial reactivity, increased pulse pressure (thought to be a surrogate for increased vascular stiffness), factor V Leiden mutation, hyperhomocysteinemia, and elevated fibrinogen.

Estrogen increases fibrinogen levels, explaining the increased risk of vascular thrombosis associated with exogenous estrogen therapy. Although several members of the coagulation and fibrinolysis cascade have been proposed as possible risk factors, most recently through genetic polymorphisms, current data support a role for these factors as modifiers of risk rather than causal.

Whether targeting any of these emerging risk factors for intervention produces a reduction in cardiovascular events is unknown.

ACUTE CORONARY SYNDROMES

The term “acute coronary syndrome” is used to describe a spectrum of conditions having in common; varying degrees of rupture of vulnerable atherosclerotic plaques is the coronary arteries.

It includes the clinical entities of unstable Angina, non-ST Elevation Myocardial Infarction and ST Elevation Myocardial Infarction.

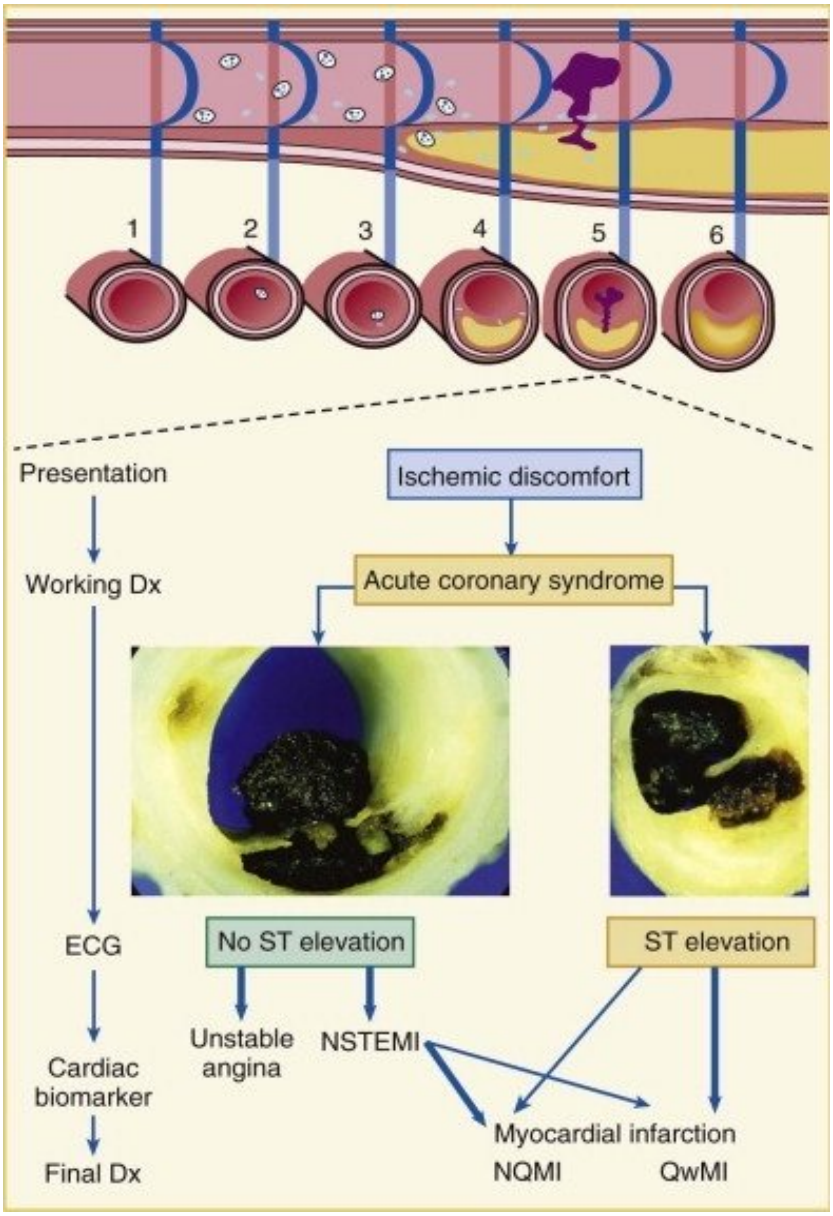


Figure 3.3 Acute coronary syndrome

Table 3.1: CORONARY ARTERY PATHOLOGY[19]

	Syndrome	Pathology
1.	Stable Angina	>75% stenoses
2.	Unstable Angina	Plaque rupture with mural thrombus, often thromboembolic
3.	Myocardial Infarction	Plaque rupture, complete thrombosis
4.	Sudden death	Severe multi vessel disease, often Plaque rupture, often thrombus or thromboembolic

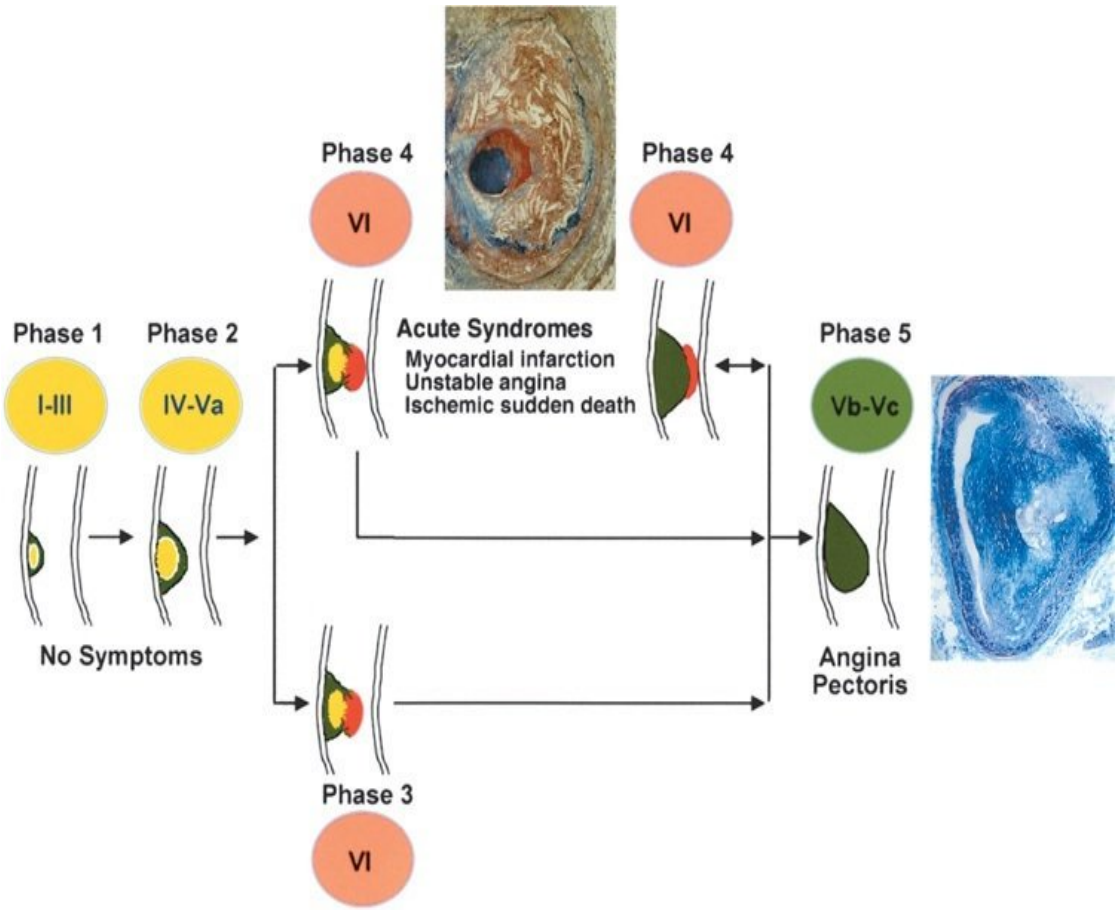


Figure 3.4 Acute syndrome

Potential outcomes of reversible and irreversible ischaemic injury

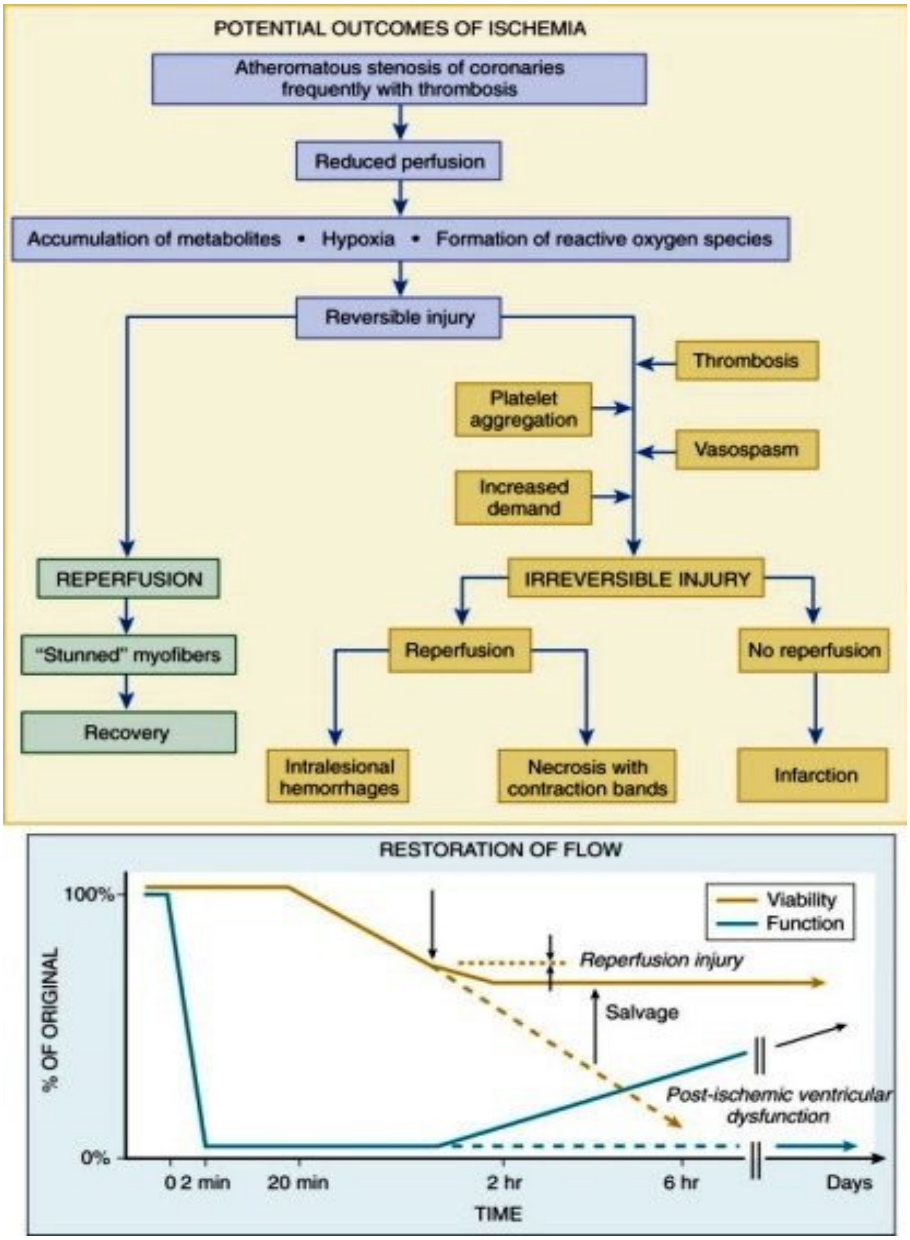


Figure 3.5 Potential outcomes of ischemia

Causes of nonatherothrombotic coronary artery disease

Arteritis:

- Infection:

  Bacterial.

  Fungal.

  Viral.

  Rickettsial.

  Spirochetal.

- Immunological

  Immune complex-mediated:lupus vasculitis, hepatitis B microscopic polyarteritis.

  Direct antibody attack-mediated:Kawasaki (antiendothelial antibodies)

  ANCA associated (mediated):Wegener's granulomatosis.

  Cell-medicated:best known is allograft organ rejection.

  Hypersensitivity reaction:drugs.

- Unknown:

  Polyarteritis nodosa (classic)

  Giant cell arteritis.

Takayasu's arteritis.

Embolism

- Infectious endocarditis.
- Nonbacterial thrombotic endocarditis (NBTE).
- Parasitic disorders.
- Vegetations on catheters or fragment of catheter: iatrogenic material.
- Tumors:

  Myxoma

  Paradoxical; embolus.

  Foreign bodies, including gas.

Occlusion of the coronary arterial orifice

- Thrombus from stalky endocarditic excrescence.
- Papillary fibroelastoma: papillary tumor of the leaflet.
- Surgical material:

  Felt pads, which can also be embolic.

  Suture material gripping the coronary artery in relation to arterioplastic operations or bypass

Dissection

- Spontaneous
- Marfan
- Hypertension.

Miscellaneous

- Congenital coronary anomalies:

  Arteriovenous malformation.

  Anomalous origin of coronary arteries.

- Fibromuscular dysplasia/hyperplasia.
- Perivascular/vascular fibrosis in hypertrophic cardiomyopathy
- Endocrine and metabolic disorders:

  Diabetes, hypothyroidism.

  Homocysteinemia, mucopolysaccharidoses.

CLINICAL FEATURES

STEMI[20]

1) Chest pain

Site	Substernal
Nature	Pressing, Squeezing, Strangling, Constricting, 'a band across the chest', 'a weight in the centre of the chest'. The patient cannot pinpoint the site of pain.
Radiation	To both the shoulders, epigastrium, back, neck, jaw, teeth. Anginal pain can be radiate in all directions, as mentioned above, but more commonly radiates to the left shoulder and ulnar aspect of the left arm.
Duration	5-15minutes
Aggravating Factors	Exertion, emotion, after a heavy meal, or exposure to cold.
Relieving Factors	Rest, nitrates.

Table 3.2

2) Often accompanied by Weakness, Sweating, Nausea, Vomiting, anxiety and Sense of impending doom.

3) May present as

- Sudden onset of breathlessness
- With or without pain
- Loss of consciousness
- A confusional state
- A sensation of profound weakness

- The appearance of arrhythmia
- Evidence of peripheral embolism
- Merely an unexplained drop in arterial pressure.

Physical signs include

1. Elevated BP, low because of cardiogenic shock
2. Basal crepitation
3. S3, S4
4. Systolic murmur of MR
5. Soft S1
6. Paradoxic S2
7. Pallor
8. Sweating, restless, in agony due to pain and tossing in the bed in an attempt to get relief
9. Pulse – rapid or slow ,regular or irregular

   Tachycardia – AWMI

   Bradycardia – IWMI
10. Pulmonary edema.

Investigations

Blood Investigations

1. Leucocytosis with polymorphonuclear reaction
2. High ESR
3. Elevated CRP

ECG changes may be normal initially, hence serial ECGs must be taken

1. ST Elevation and T wave inversion[21]
2. T wave inversion in opposite leads
3. Q wave on the ECG

Cardiac Markers

1. Cardiac – Specific Troponin T and I[22]
2. Creatinine Phosphokinase[23]
3. LDH1
4. AST
5. Myoglobin.

Enzyme levels in acute MI

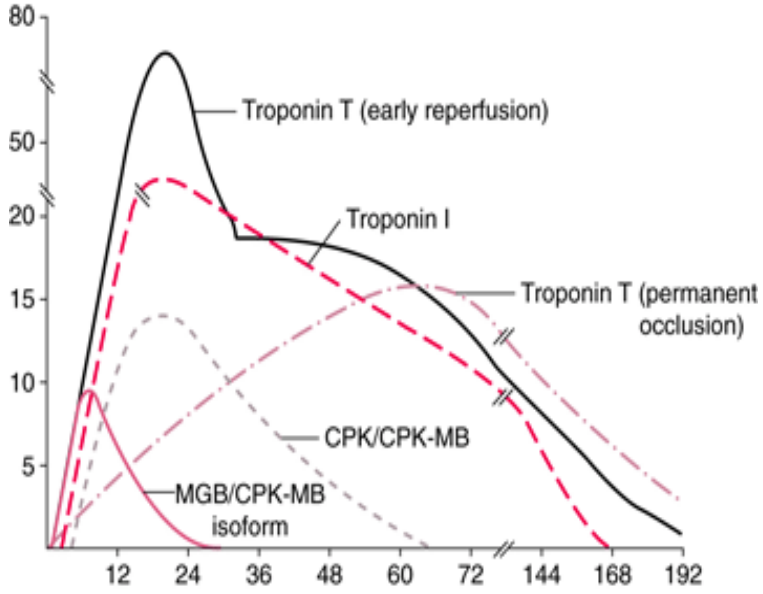


Figure 3.6 Enzyme levels according to time

Radionuclide imaging techniques

Myocardial perfusion imaging with ^{201}T1 or ^{99m} TC – sestamibi, which are distributed in proportion to myocardial blood flow and concentrated by viable myocardial, reveal a 'cold spot' (defect).

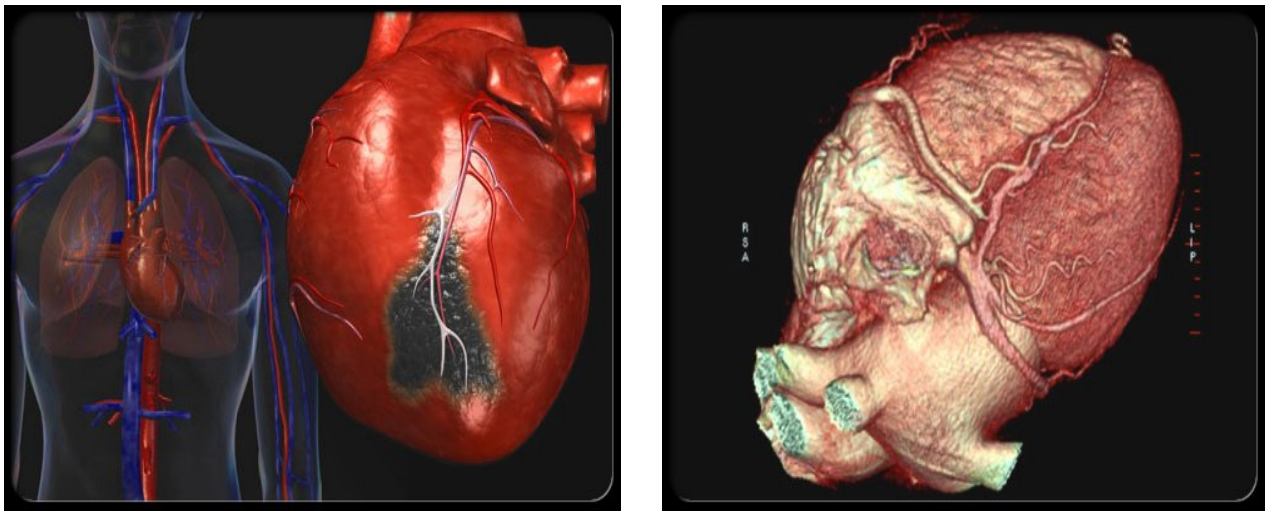


Figure 3.7 Block shows in Infarcted area and 18 sliced CT angiogram

^{99m}TC- labeled red blood cells frequently demonstrates wall motion disorders and reduction in the ventricular ejection fraction.

Myocardial infarction can be detected accurately with high resolution cardiac MRI using (gadolinium) a technique referred to as late enhancement.

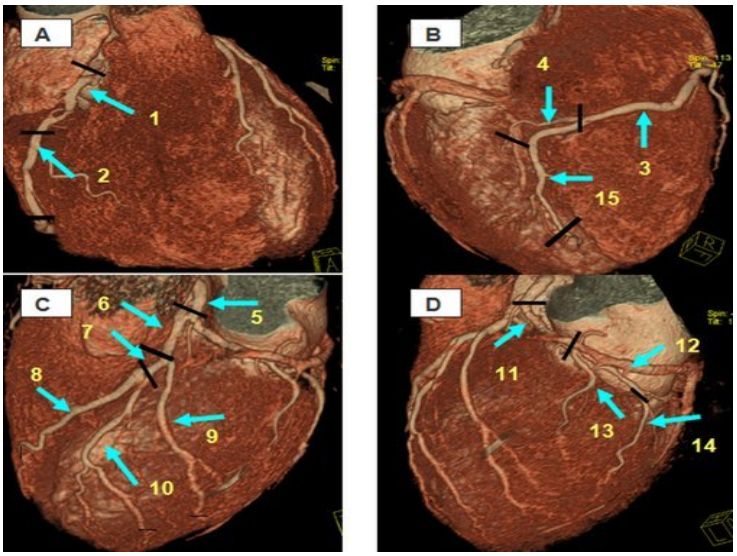


Figure 3.8 Shows in 64 slice computed tomography in coronary angiogram

a) CPK-MB	This cardiac isoenzyme starts rising within 4-6 his after development of acute MI, peaks during the 2nd day (4 fold risk) and disappears in 2-3days
b) ASI	Starts rising on the 1st day, peaks in 2-3days (3 fold rise) and disappears by 3rd day.
c) LDH1	Starts rising by second day, peaks around 3-4days (3 fold rise) and disappears in 10 days.
d) Troponin T	Cardiac Troponin T is a regulatory contractile protein hot normally found in blood. Its detection in the circulation has been shown to be a sensitive and specific marker for myocardial cell damage. Troponin T and I reach a reliable diagnostic level in plasma by 12-16 hrs, maximal activity by 24-32 hrs, returns to normal in 10-12 days. Troponin I :0-0.4 ng/ml Cardiac troponin are detected in the serum by using monoclonal antibodies. These antibodies have negligible cross reactivity to skeletal muscle. Cardiac troponin I and T start to rise within 3-4 hours after myocardial infarction and remain raised for 4-10 days.

Chest X-ray

Signs of heart failure or pulmonary edema.

Cardiac Imaging

Echo

Abnormalities of wall motion on two-dimensional echocardiography are almost universally present. LV function estimation is assessed. Echo may also identify the presence of RV infarction, ventricular aneurysm, pericardial effusion, LV thrombus.

Doppler echo detects – VSD, MR

Management

1. Asses the vital signs
2. Establish an IV line
3. Connect the patient to a cardiac monitor
4. Measure O_2 saturation in breathless patients.

1) Start O_2 using a nasal canula (4-6l/min)

Especially in pulmonary edema or oxygen saturation <90% continue for 4-6 hrs.

2) Aspirin (325mg) is given to the patient, to be chewed and swallowed continue 150-325mg / day indefinitely.

3) Inj.Morphine – for Analgesia 5 mg IV stat and 2 mg IV can be repeated every 10 minutes till adequate analgesia is achieved (Maximum dose of 16mg)

Caution: in inferior wall MI as it may precipitate heart blocks Inj.Pethidine 50-100 mg IV with Inj.Phenargan 12.5-25 mg IV is an alternative analgesia.

4) a) Sublingual nitroglycerine

- 0.4 mg can be repeated every 15 minutes maximum of 3 tablets
- If the pain is not relieved with adequate sublingual Nitroglycerine tablets and morphine injection, start nitroglycerine drip.

b) Nitroglycerine drip

Indications

- For 24-489 hours in case of persistent angina
- Large AMI
- MI with CCF or hypertension .

10 µgram/min to increase to 200 µgram/min for pain relief.Titrate dose to pain relief with monitoring of blood pressure.

CI

Hypotension- SBP less than 90mmHZ change over to oral nitrates when pain subsides.

5) Thrombolysis with streptokinase (to decide and start at the earliest, ideally within 30 minutes of chest pain/entry to emergency department)

   If there are no contraindications and the BT, CT, PT, APTT and platelet count are normal.

   - Best response it used within 6 hrs of onset
   - Can be used upto 12-24 hrs.

Indications

- Typical chest pain
- ECG/enzyme evidence
- <12 hrs from onset of pain
- Age less than 75 yrs.

Major contraindication for streptokinase[24]

- Stroke within the previous 12 months
- Severe hypertension BP >180/110mmHg
- Bleeding disorders and patient on anticoagulants.
- Active peptic ulcer
- Surgery within the previous one month
- Recent aggressive cardiac resuscitation

- Previous treatment with SK

Use of SK

- 1.5 million units dissolved in 100 ml of NS to run in one hour
- 750000 units in the first 20 minutes as IV infusion
- 750000 units in the next 40 minutes as IV infusion
- Give 100 mg of hydrocortisone and 25 mg pheniramine meleate IV before giving SK to present minor allergic reaction
- If BP falls, give volume expanders like saline.

6) If pain persists after thrombolysis consider urgent PTCA or CABG.

7) Inj.Heparin

   - To prevent clot formation and embolization in cases of large MI/AI
   - LV thrombus – stabilize the clot
   - Following thrombolysis/PTCA/CABG.

   UFH

   - gives 6 hrs after the SK to maintain APTT Around 1.5 to 2 times the control for 5-7days
   - 5000 IU IVstat followed by 1000 U/hour IV alternatively

LMWH

- Given 5-7 days Enoxaparin 1 mg/kg/BD
- No need to monitor APTT but it is expensive

8) Beta blockers

Indications

1. All patients with 12 hrs of MI with no major contraindication to this drug
2. Patients with recurring or ongoing chest pain
3. Arrhythmias such as AF/VF
    - Metoprolol 5mg IV stat and repeat every 5 minutes if needed till 15 mgs.
    - Use oral beta-blockers indefinitely'
    - Metoprolol 50mg orally BD.

Use with caution

1. IWMI
2. SBP < 95mm/Hg
3. HR < 55/min
4. Severe LVD
5. Heart blocks

6. Severe COPD/Asthma

9) **ACE** inhibitors

If cardiac failure / LVD/

- Enalapril 5-20mg
- Ramipril 2.5-20mg

Avoid in hypotension /renal failure/ hyperkalemia.

10) Calcium channel blockers

- Used only when beta-blockers are contraindicated and there is ongoing chest pain.
- To control ventricular rate in AF
- Avoid in CCF and LVD
- Diltiazem – bolus 0.25 mg/kg IV over 2-5 min for arrhythmias
- Infusion dose is 5-15mg / hour. 250mg in 250 ml = 1ml. infusion rate 5-15ml/hour or microdrops/min or Diltizem PO 30-60mg 8^{th} hourly.

11) Amiodarone

- For atrial and ventricular arrhythmia.
- Bolus – 150mg in 20ml 5% D over 5-15 minutes.
- Infusion – Inj.Amiodarone 1amp = 3ml = 150mg

- 6amp in 500ml of 5% D slow loading dose 1mg/min.

Supportive Treatment

1. Reassurance
2. Complete Bed rest for 12-24 hrs if no complications
3. Movements of legs in bed and deep breathing exercises
4. Avoid straining or valsalva for any reason including defecation and passing urine
5. Laxative to be given to avoid straining during defaecation
6. Toilet facilities near bedside
7. Sedatives like Diazepam 5-10mg or alprazolam 0.5mg should be given at night and a calm and quiet atmosphere to be maintained
8. Diet – It patient in stable soft diet, small frequent feeds are started after 6 hrs of MI.
9. Treat Hyperlipidemia
    - If LDL >100mg/dl or
    - HDL <35mg/dl.
10. Specific treatment of complication
    - Severe hypotension
    - Pulmonary edema
    - Arrhythmias

    } Should be treated

Refer for cardiac pacing if there is

1. Asystolic
2. Symptomatic bradycardia
3. BBB or bifascicular blocks or complete heart block.

While waiting for cardiac pacing the following drugs can be tried

- Inj.Atropine 1mg IV, total 3 doses
- Inj.Deriphylline 1 amp
- Inj.Isoprenaline 2mg in 500ml NS/5% D.

NSTEM1 AND UNSTABLE ANGINA ARE CLOSELY RELATED IN PATHOGENESIS AND CLINICAL PRESENTATION.

UNSTABLE ANGINA

Symptoms

1. Angina at rest
2. Recent onset severe angina
3. Progressing severity of angina
4. Post infarction angina

ECG

1. May show ST depression and

2. T wave inversion
3. NO ST elevation /Q wave
4. Normal cardiac enzymes

Treatment

1) Aspirin 325mg oral stat and daily. If not tolerated, ticlodipine 250mg BD monitor for neutropenia.
2) **Heparin** – unfractionated

   1000 IU/hr, as an infusion with monitoring APTT (Or)

   LMW Heparin – given for 5 days

   1) Enoxaparin 1mg/kg SC BD
   2) Dalteparin 100 IU/kg SC BD
   3) Nandopanin 50 IU/kg SC BD
   4) Reviparine 3500-6300 U SC BD
   5) Fondaparinux - OD

3) Beta blockers

   - Atenolol 50-100mg
   - Metoprolol 50-100mg

   } 12^{th} hrly

- Watch for BP fall and bradycardia

4) Nitrates

- NTG IV starting from 10-30µg/mt upto 200-400 µg/min. Titrate the dose to relieve pain
- Watch for hypotension <90mmHg
- In hypertensives do not lower below 120mmHg systolic
- If Pt has mild pain, oral or S/L glyceryl trinitrate 0.5mg every 3 minutes till the pain subsides C maximum 3mg in (hr).

5) Calcium channel blockers

- Used only if beta blockers are contra indicated,
- Avoid short acting drugs like nifedipine
- Diltiazem 30 to 60 mg TID can be given.

6) Treat Associated Aggravating factors like anemia, stress, fever, tachyarrhythmia

- Detect and treat risk factors like DM, SHT, Lipid abnormalities
- Advice to stop smoking
- If pain subsides, Treadmill test on drugs is indicated, after the patient in stable.
- Depending upon the outcome revascularistion can be planned for high risk patients.

- Refractory Angina – Refer for emergency PTCA/CABG[24] .

Complications

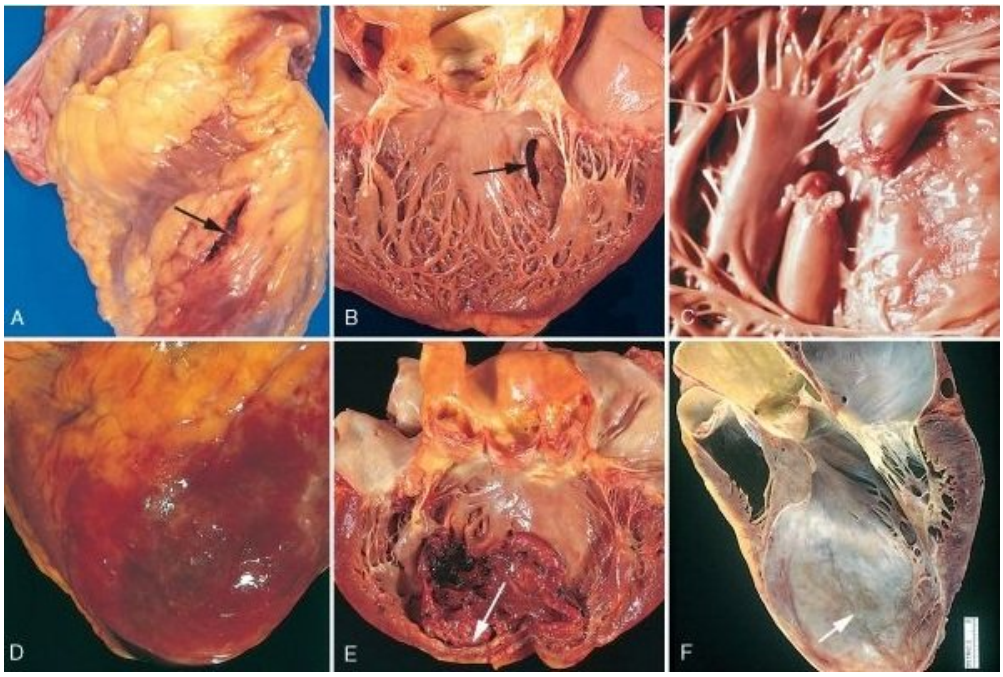


Figure 3.9 Complications

A, Anterior myocardial rupture in an acute infarct *(arrow)*. **B**, Rupture of the ventricular septum *(arrow)*. **C**, Complete rupture of a necrotic papillary muscle. **D**, Fibrinous pericarditis, showing a dark, roughened epicardial surface overlying an acute infarct. **E**, Early expansion of anteroapical infarct with wall thinning *(arrow)* and mural thrombus. **F**, Large apical left ventricular aneurysm. The left ventricle is on the *right* in this apical four-chamber view of the heart.

1. Arrhythmias

2. Heart failure

3. Cardiogenic shock

4. Sudden death

5. Infarction of papillary muscle of mitral value

6. Rupture of Interventricular septum leading to VSD

7. Thrombosis in LV causing cerebral embolism

8. Rupture of ventricle into pericardial sac causing cardiac tamponade

9. Deep vein thrombosis is legs causing pulmonary embolism

10. Pericarditis during massive infarction

11. Aneurysm of ventricle with thrombosis and thrombo embolic phenomenon

12. Dressler's syndrome.

Table 3.3: Evaluation and interventions in relation to different components of a multifactorial rehabilitation program		
	Evaluation	**Intervention**
Patient assessment	Medical history Physical examination	Compose patient care program.
Nutritional counseling	Obtain estimate of daily food intake Assess eating habits	Prescribe dietary modifications. Individualize eating plan. Educate and counsel patient (and family).
Smoking cessation	Document smoking status Determine readiness to change	Provide formal smoking cessation program Update status at each visit
Weight management	Measure weight height, and circumference. Calculate BMI	In patients with BMI>25 establish reasonable short-term and long-term weight goals. Develop a combined diet, exercise and behavioral program.
Exercise training	Obtain an exercise test	Develop a documented individualized exercise prescription for aerobic resistance training.
Psychosocial management	Use interview and/or standardized measurement tools to identify psychosocial distress	Offer individual education and counseling. Develop supportive rehabilitation environment to enhance social support Cooperate with appropriate mental health specialist.
Discharge therapy	Evaluate relevant long-term therapy with aspirin, beta blockers, and ACE inhibitors.	Monitor dose adjustments and side effects (ECG, kidney function)
Lipid management	Obtain fasting measures of total cholesterol, HDL, LDL and TG. Repeat lipid profiles 4-6 weeks after hospitalization and 2 months after changes in therapy.	Provide nutritional counseling and add drug treatment until: LDL <100 mg/dl (2.8 mmol/l), HDL >35 mg/dl (1.0 mmol/l), TG <200 mg/dl (2.5 mmol/l).
Hypertension and diabetes management	Measure of resting BP on at least 2 visits. Identify diabetic subjects. Obtain fasting plasma glucose in all patients and HbA l c in diabetic patients to monitor therapy.	Continue assessment and optimize treatment until:BP <140/90 mmHg [18.7/12 kPa] or BP <130/85 mmHg [17.3/11.3 kPa] (diabetics). Continue monitoring and optimize diet, exercise, and oral hypoglycemic agents or insulin until near normalization of glycemic control with HbA l c <7.0%.

The DASH Diet:

Food Group	Daily Serving	Examples and Notes
Grains	7-8	Whole wheat bread, oatmeal, popcorn
Vegetables	4-5	Tomatoes, potatoes, carrots, beans, peas, squash, spinach
Fruits	4-5	Apricots, bananas, grapes, oranges, grapefruit, melons
Low-fat or fat-free dairy foods	2-3	Fat-free (skim)/low-fat (1%) milk, fat-free/low-fat yogurt, fat-free/low-fat cheese
Meats, poultry, fish	2	Select only lean meats. Trim away fats. Broil, roast or boil. No frying. Remove skin from poultry.
Nuts, seeds, dry beans	4-5/week	Almonds, peanuts, walnuts, sunflower seeds, soybeans, lentils
Fats and oils	2-3/	Soft margarines, low-fat mayonnaise, vegetable oil (olive, corn, canola, or safflower).
Sweets	5/week	Maple syrup, sugar, jelly, jam, hard candy, sorbet

4. MATERIALS AND METHODS

PLACE OF THE STUDY

- The study was conducted at Government Coimbatore Medical College hospital, Coimbatore (Tertiary Care Centre).

PERIOD OF STUDY

- September 2010 to June 2011

DESIGN

- Prospective cross sectional study.

METHODOLOGY

- Hundred female patients admitted with symptoms and signs and ECG changes suggestive of CAD with Biochemical markers taken as cases.

INCLUSION CRITERIA

1. Patients above 40 years
2. Hypertension
3. Diabetes mellitus
4. Dyslipidemia

EXCLUSION CRITERIA

1. Congenital Heart disease.
2. Rheumatic Heart disease.

3. Structural Heart disease.
4. Electrical abnormalities.

- Admitted 100 female patients were examined and detailed history was taken about the patients menstrual history, sedentary habits, Dietary habits, Smoking habits, h/o Alcohol intake, h/o OCP intake, Family h/o and systemic hypertension and Type 2 DM and others risk factor analysis was made.

- Baseline investigations like complete blood count, urine R/E, renal function test, ECG, Chest X-ray, Blood pressure monitoring was done in all study subjects.

- Hypertension was considered by documented history of hypertension/medication or with BP >140/90 mmHg, during the present visit.

- Diabetes mellitus was considered by documented history. Fasting blood Sugar and Postprandial Blood Sugar values were evaluated.

1. FBS- 126 & Above
2. PPBS-160 & Above

} Taken as criteria for DM.

- Clinical Symptoms like chest pain, Nausea, Vomiting, Dysponea, Syncope Radiation of the pain were all taken into account.

- Signs like hypotension, hypertension, raised JVP, S3, S4, Lung Crepitations, where were all looked for.

- Total cholesterol, LDL, HDL, Triglycerides levels identified and taken into the study.

- Patients were grouped into four categories according to their age as 40-50 yrs, 50-60 yrs, 60-70 yrs, above 70 yrs.

- Body mass Index was calculated by wt(kg)/ht(m^2) in all patients. Waist/Hip ratio calculated in all patients.

Killip Score was assigned according to the clinical presentation.

Killip Classification

Class	Features
I	No heart failures
II	Mild to moderate heart failure (S3; rales no more than half way to the back)
III	Severe heart failure (Pulmonary edema)
IV	Cardiogenic shock

Table 4.1

- Electrocardiogram was performed in all study subjects

- Echocardiogram was performed in all study subjects

- Troponin study was done in NSTEMI and unstable angina patients.

Types of ECG Abnormalities Recorded

- ST Elevation MI

- Non ST Elevation MI

- Unstable Angina

ECG PATTERNS

- Extensive anterior Wall MI – L1, avL and Precordial leads.
- Anteroseptal wall MI –V1 to V4.
- Anterolateral wall MI –L1, avL, V4, V5, V6.
- Apical wall MI –V5, V6.
- Inferior wall MI –LII, LIII, avF.
- Interolateral wall MI –LII, LIII avF, V5, V6.
- RVMI –V1 and V4 R
- Postenior wall MI –Right V1-V3 and especially V2 – respect the universe charge.

BMI

CLASSIFICATION	BMI
Underweight	<18.50
Normal range	18.50 – 24.99
Overweight	≥ 25.00
Pre – obese	25.00 – 29.00
Obese Class I	30.00 – 34.99
Class II	35.00 – 39.99
Class III	≥ 40.00

WAIST HIP RATIO

More than 0.85 considered as risk for metabolic complications.

LIPID PROFILES

Total cholesterol (mg%)

<200	Desirable
200-239	Borderline high
>240	High

LDL cholesterol (mg%)

<100	Optimal
100-129	Near optimal/above
160-180	High
≥ 190	Very high.

HDL (mg%)

<40	Low
≥ 60	Borderline high

TGL (mg%)

<150	N
150-199	Borderline high
200-499	High
≥ 500	Very high.

STATISTICAL METHODS EMPLOYED

Following statistical methods were employed in the present study.

1. Chi-square test
2. Independent samples‘t’ test
3. One-Way ANOVA.

5. RESULTS AND ANALYSIS

1. **AGE WISE DISTRIBUTION**

   100 patients were studied in this study grouped into four category.

   - Between 40-50 yrs
   - Between 50-60 yrs
   - Between 60-70 yrs
   - Above 70 yrs

Age	Frequency (No of Patients)
Between 40-50 yrs	17
Between 50-60 yrs	24
Between 60-70 yrs	33
Above 70 yrs	26
Total	100 patients

2. **BMI (wt(kg)/ht(m^2))**

   Observations

   In this study

   - Healthy – 26 patients
   - Overweight – 51 patients
   - Obese – 23 patients

Age wise distribution of BMI

Age	Healthy	Overweight	Obese	Total
Between 40-50 yrs	5	9	3	17
Between 50-60 yrs	6	13	5	27
Between 60-70 yrs	10	14	9	33
Above 70 yrs	5	15	6	26
Total	26	51	23	100

3. WAIST HIP RATIO

- 0.85 considered as significant
- 81% (81 out of 100 patients presented with >0.85 WHR.
- 19% (19 out of 100 patients) presented with <0.85 WHR.

4. MENSTRUATION

In this study 7% (7 out of 100) patients were menstruating women.

5. SEDENTARY HABITS

In this study 89% (89 out of 100) patients reported to have sedentary habits.

Age	Sedentary Habits		Total
	No	Yes	
Between 40-50 yrs	4	3	17
Between 50-60 yrs	1	23	24
Between 60-70 yrs	4	29	33
Above 70 yrs	2	24	26
Total	11	89	100

BMI * SED

Crosstab

			SED		
			No	Yes	Total
BMI	Healthy	Count	6	20	26
		% within BMI	23.1%	76.9%	100.0%
		% within SED	54.5%	22.5%	26.0%
	Over weight	Count	4	47	51
		% within BMI	7.8%	92.2%	100.0%
		% within SED	36.4%	52.8%	51.0%
	Obese	Count	1	22	23
		% within BMI	4.3%	95.7%	100.0%
		% within SED	9.1%	24.7%	23.0%
Total		Count	11	89	100
		% within BMI	11.0%	89.0%	100.0%
		% within SED	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	5.432[a]	2	.066
Likelihood Ratio	4.944	2	.084
Linear-by-Linear Association	4.509	1	.034
N of Valid Cases	100		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is 2.53.

BMI and Sedentary Habits

BMI	Sedentary Habits		Total
	No	Yes	
Healthy	6	20	26
Overweight	4	47	51
Obese	1	22	23
Total	11	89	100

Chest pain and Sedentary Habits

Chest Pain	Sedentary Habits		Total
	No	Yes	
No	1	11	12
Yes	10	78	88
Total	11	89	100

Age * Chest Pain

Crosstab

			Chest Pain		
			No	Yes	Total
Age	Between 40 - 50 years	Count	3	14	17
		% within Age	17.6%	82.4%	100.0%
		% within Chest Pain	25.0%	15.9%	17.0%
	Between 50 - 60 years	Count	3	21	24
		% within Age	12.5%	87.5%	100.0%
		% within Chest Pain	25.0%	23.9%	24.0%
	Between 60 -70 years	Count	4	29	33
		% within Age	12.1%	87.9%	100.0%
		% within Chest Pain	33.3%	33.0%	33.0%
	Above 70 years	Count	2	24	26
		% within Age	7.7%	92.3%	100.0%
		% within Chest Pain	16.7%	27.3%	26.0%
Total		Count	12	88	100
		% within Age	12.0%	88.0%	100.0%
		% within Chest Pain	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.976[a]	3	.807
Likelihood Ratio	.978	3	.807
Linear-by-Linear Association	.869	1	.351
N of Valid Cases	100		

a. 4 cells (50.0%) have expected count less than 5. The minimum expected count is 2.04.

6. No smoking habits patients were studied. 2% (2out of 100 patients) reported to have habits of alcohol intake.

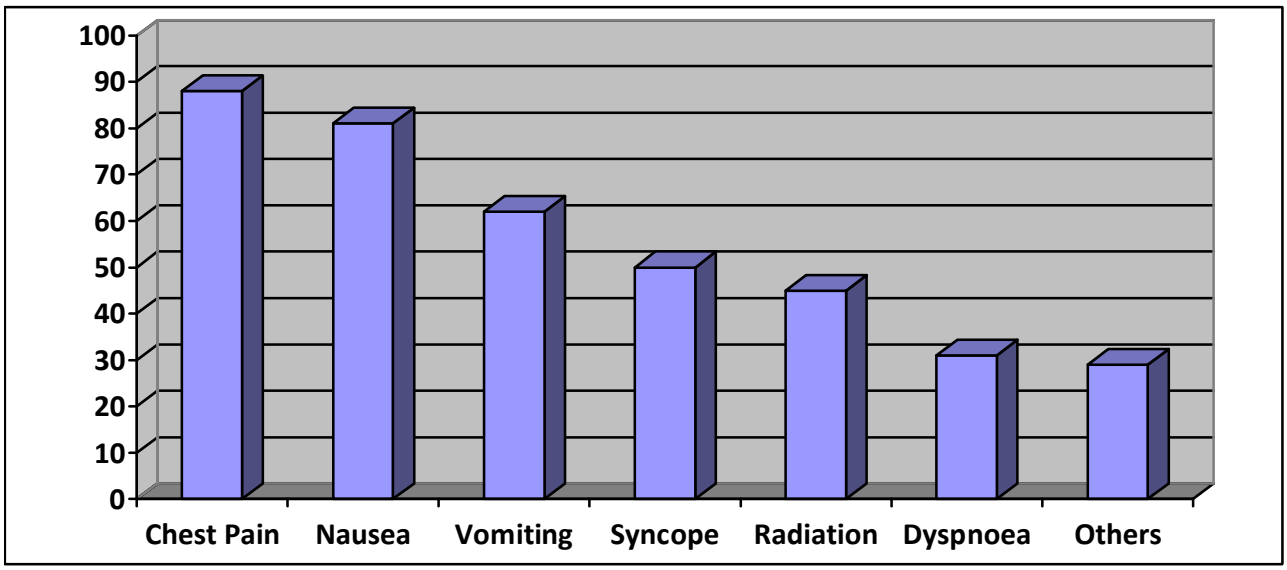
7. **History**

Oral contraception intake was questioned and found that 40% of the patient gave the history in this study (40 out of 100 patients).

8. **Clinical Symptoms**

- Chest pain is the most common presenting symptom.
- 88% (88 out of the 100 patients) presented with chest pains.

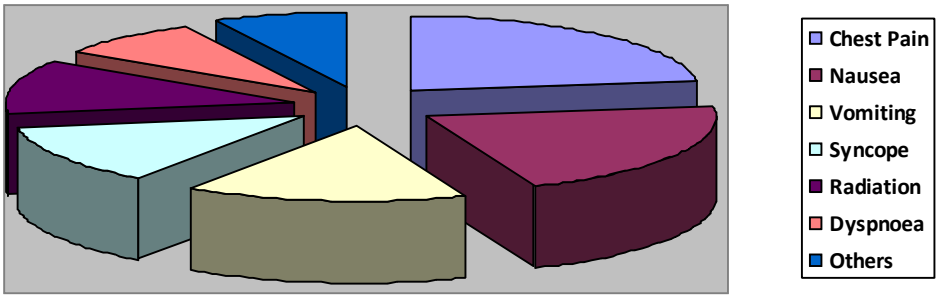
Clinical Symptoms



Symptoms

- Chest pain – 88%
- Nausea – 81%
- Vomiting – 62%
- Syncope – 50%
- Radiation – 45%
- Dyspnoea – 31%
- Other (Palpitation, epigastric pain) – 29%

Clinical Symptoms

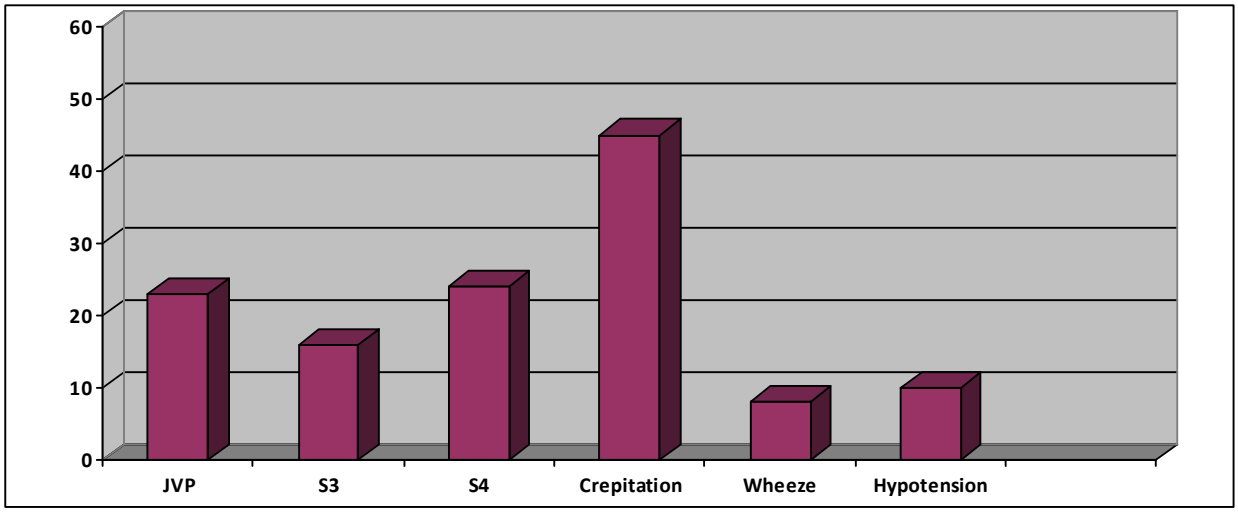


9. Clinical signs

- Lung basal Crepitation is the most common presentation of clinical sign.

Signs	No of Cases (Total = 100 patients)
JVP	23
S3	16
S4	24
Crepitations	45
Wheeze	8
Hypotension	10

Clinical Signs

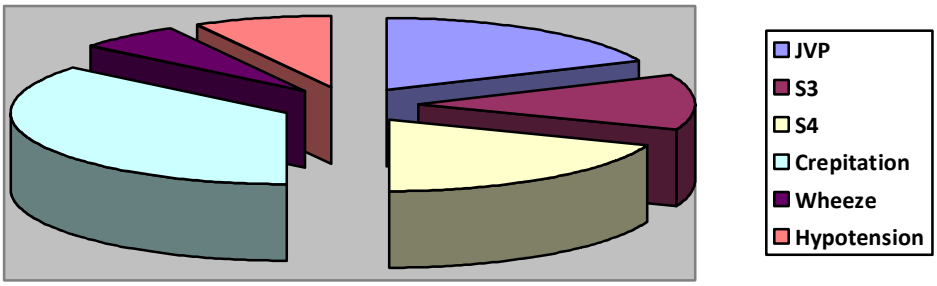


Signs

- JVP – 23%
- S3 – 16%

- S4 – 24%
- Crepitation – 45%
- Wheeze – 8%
- Hypotension – 16%

Clinical Signs



10. Type 2DM

In this study

- 45% (45 out of 100 patients) presented with elevated fasting blood sugar level.
- 50% (50 out of 100 patients) presented with elevated post prandial glucose level.

Age and Fasting Blood Sugar

Age	Fasting Blood Sugar		Total
	Normal	Abnormal	
Between 40-50 yrs	8	9	17
Between 50-60 yrs	17	7	24
Between 60-70 yrs	16	17	33
Above 70 yrs	14	12	26
Total	55	45	100

Age and Postprandial Blood Sugar

Age	Postprandial Blood Sugar		Total
	Normal	Abnormal	
Between 40-50 yrs	6	11	17
Between 50-60 yrs	14	10	24
Between 60-70 yrs	18	15	33
Above 70 yrs	12	14	26
Total	50	50	100

Chest pain and Fasting Blood Sugar

Chest (yes/no)	Fasting Blood Sugar		Total
	Normal	Abnormal	
No	6	6	12
Yes	49	39	88
Total	55	45	100

Chest Pain * Fasting Blood Sugar (Diabetes Mellitus)

Crosstab

			Fasting Blood Sugar (Diabetes Mellitus)		
			Normal	Abnormal	Total
Chest Pain	No	Count	6	6	12
		% within Chest Pain	50.0%	50.0%	100.0%
		% within Fasting Blood Sugar (Diabetes Mellitus)	10.9%	13.3%	12.0%
	Yes	Count	49	39	88
		% within Chest Pain	55.7%	44.3%	100.0%
		% within Fasting Blood Sugar (Diabetes Mellitus)	89.1%	86.7%	88.0%
Total		Count	55	45	100
		% within Chest Pain	55.0%	45.0%	100.0%
		% within Fasting Blood Sugar (Diabetes Mellitus)	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.138[b]	1	.711		
Continuity Correction [a]	.004	1	.951		
Likelihood Ratio	.137	1	.711		
Fisher's Exact Test				.764	.472
Linear-by-Linear Association	.136	1	.712		
N of Valid Cases	100				

a. Computed only for a 2x2 table

b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 5. 40.

Chest pain and Postprandial Blood Sugar

Chest (yes/no)	Postprandial Blood Sugar		Total
	Normal	Abnormal	
No	7	5	12
Yes	43	45	88
Total	50	50	100

Chest Pain * Post Prandial

Crosstab

			Post Prandial		
			Normal	Abnormal	Total
Chest Pain	No	Count	7	5	12
		% within Chest Pain	58.3%	41.7%	100.0%
		% within Post Prandial	14.0%	10.0%	12.0%
	Yes	Count	43	45	88
		% within Chest Pain	48.9%	51.1%	100.0%
		% within Post Prandial	86.0%	90.0%	88.0%
Total		Count	50	50	100
		% within Chest Pain	50.0%	50.0%	100.0%
		% within Post Prandial	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.379[b]	1	.538		
Continuity Correction[a]	.095	1	.758		
Likelihood Ratio	.380	1	.537		
Fisher's Exact Test				.760	.380
Linear-by-Linear Association	.375	1	.540		
N of Valid Cases	100				

a. Computed only for a 2x2 table

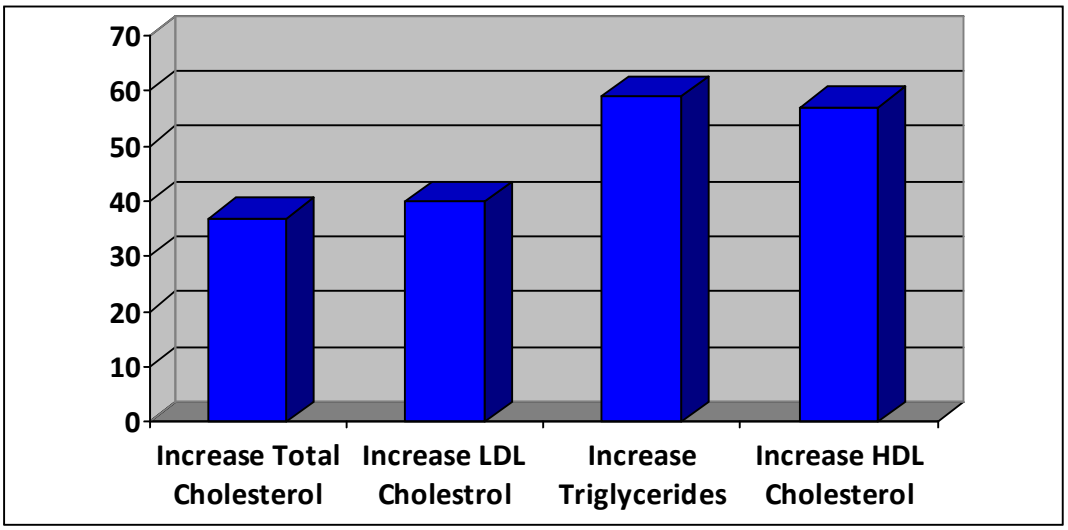
b. 0 cells (.0%) have expected count less than 5. The minimum expected count is 6.00.

11. Systemic Hypertension

In this study 25% (25 out of 100) patients presented with High blood pressure.

12. Lipid abnormalities

- 37% (37 out of 100 patients) found to have high levels of cholesterol.
- 57% (57 out of 100 patients) found to have HDL <50 m/dl.
- 40% (40 out of 100 patients) found to have high LDL cholesterol >130 mg/dl.
- 59% (59 out of 100 patients) found to have high levels of triglycerides.



BMI * Total Cholesterol

Crosstab

			Total Cholesterol		
			Normal	Abnormal	Total
BMI	Healthy	Count	19	7	26
		% within BMI	73.1%	26.9%	100.0%
		% within Total Cholesterol	30.2%	18.9%	26.0%
	Over weight	Count	31	20	51
		% within BMI	60.8%	39.2%	100.0%
		% within Total Cholesterol	49.2%	54.1%	51.0%
	Obese	Count	13	10	23
		% within BMI	56.5%	43.5%	100.0%
		% within Total Cholesterol	20.6%	27.0%	23.0%
Total		Count	63	37	100
		% within BMI	63.0%	37.0%	100.0%
		% within Total Cholesterol	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.654[a]	2	.437
Likelihood Ratio	1.699	2	.428
Linear-by-Linear Association	1.467	1	.226
N of Valid Cases	100		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 8.51.

BMI * Triglycerides

Crosstab

			Triglycerides		
			Normal	Abnormal	Total
BMI	Healthy	Count	11	15	26
		% within BMI	42.3%	57.7%	100.0%
		% within Triglycerides	26.8%	25.4%	26.0%
	Over weight	Count	22	29	51
		% within BMI	43.1%	56.9%	100.0%
		% within Triglycerides	53.7%	49.2%	51.0%
	Obese	Count	8	15	23
		% within BMI	34.8%	65.2%	100.0%
		% within Triglycerides	19.5%	25.4%	23.0%
Total		Count	41	59	100
		% within BMI	41.0%	59.0%	100.0%
		% within Triglycerides	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.482[a]	2	.786
Likelihood Ratio	.489	2	.783
Linear-by-Linear Association	.262	1	.609
N of Valid Cases	100		

a. 0 cells (.0%) have expected count less than 5. The minimum expected count is 9.43.

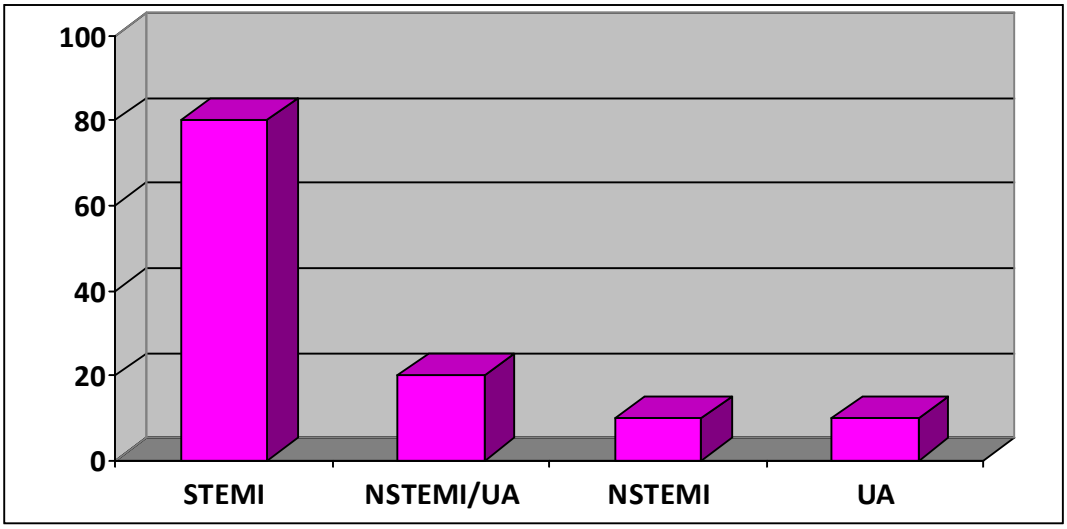
13. ECG was performed in all patients

Types of Abnormalities

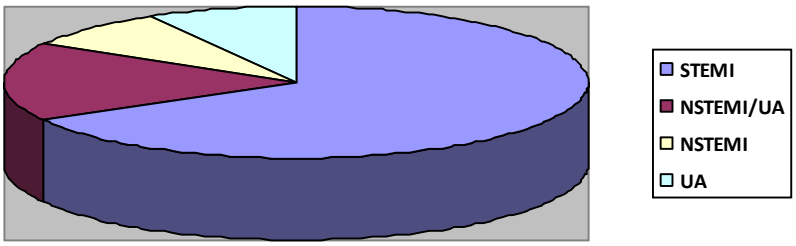
- ST Elevation MI – 80%
- Non ST Elevation MI – 10%
- Unstable Angina – 10%

Types of MI	No of Patients
ASMI	32
AWMI	20
IWMI	16
IWMI + RVMI	9
IWMI + RVMI + PWMI	2
ALMI	1
Total	80

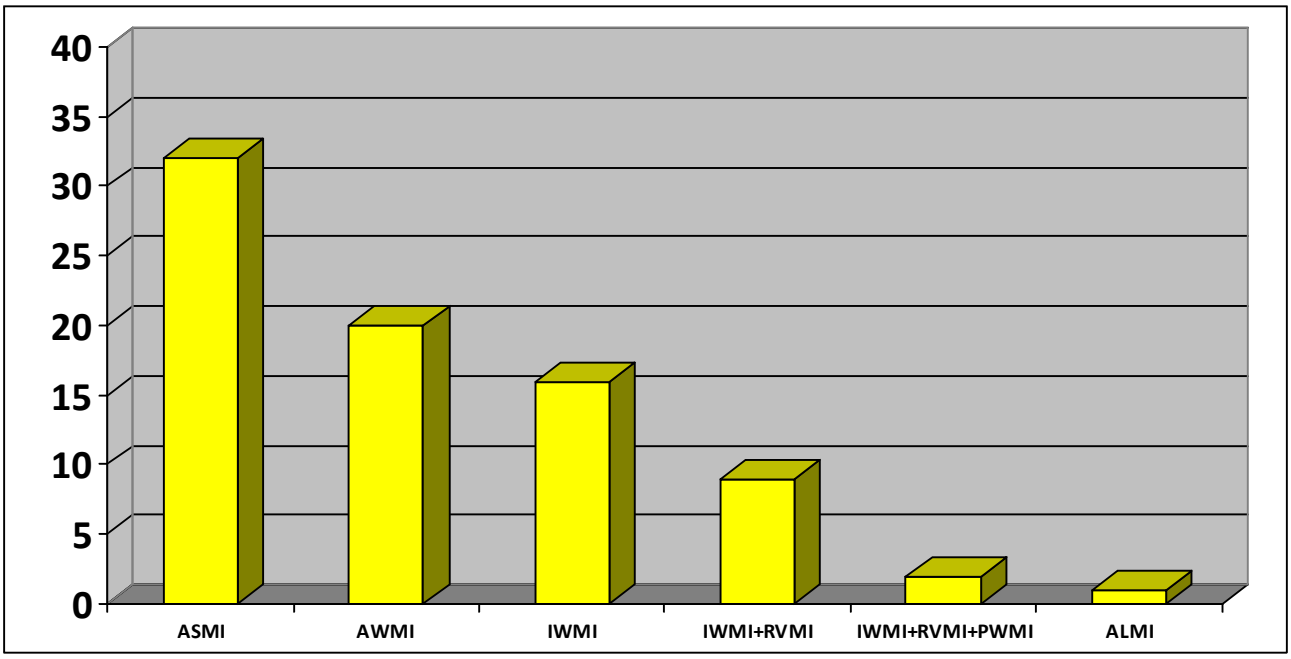
ECG Features of CAD



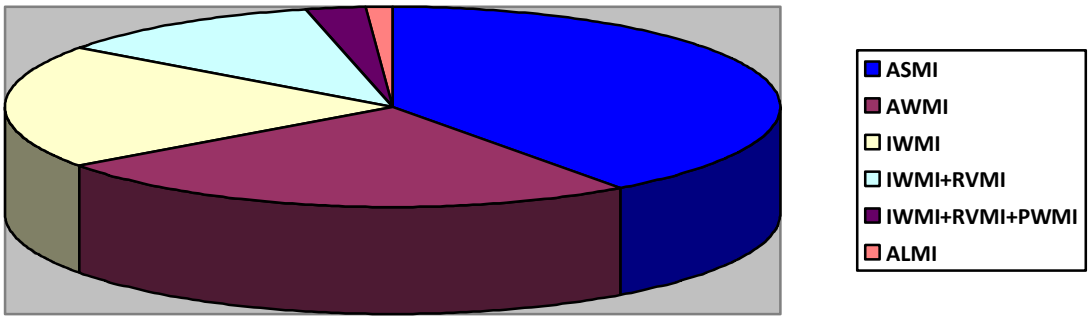
ECG Features of CAD



ECG Features of STEMI



ECG Features of STEMI



14. Troponin

- Cardiac markers study done in NSTEMI patients.
- 50% (10 out of 20 patients) found to have specific cardiac markers.

15. Echo was performed in all study groups.

16. CRP was performed in all study groups and 71% found to have positive (Elevated) CRP (71 out of 100 patients).

17.

Age	**Thrombolysed**		**Total**
	No	**Yes**	
Between 40-50 yrs	11	6	17
Between 50-60 yrs	18	6	24
Between 60-70 yrs	25	8	33
Above 70 yrs	25	1	26
Total	79	21	100

6. DISCUSSION

Coronary artery diseases are major causes of mortality and disease in the Indian subcontinent with more than 25% deaths. It has been predicted that these diseases will increase rapidly in India and this country will be host to more than half the cases of heart diseases in the world within the next 15 yrs.

Women constitute 48% of the total population in India. However due to inadequate perception and attention, coronary heart disease remains a formidable health problem in women.

- Women represent 60% of those over the age of 65 years in the United States (US) and more women than men have died of cardiovascular disease (CVD) since 1984.
- Among women, the lifetime risk of death from CAD is more than 10-fold greater than that from breast cancer
- Since 1960, life expectancy in India has increased by 20 years to 61 years of age.
    - In our study 33% were among age group between 60-70 yrs. Coronary artery disease mortality among women gradually increases with age and increase in the risk of coronary artery disease is related to a higher incidence of hypertension, diabetes, obesity and dyslipidemia.
- From 1960 to 1995, the prevalence of CAD in adults increased from 3% to 10% in urban Indians and from 2% to 4% in rural Indians, with women having rates similar to men.
- In 1990, there were 783 000 deaths due to CAD in India and this is projected to double by the year 2015, primarily due to affluence and urbanization.

- In the FHS, only 17% of women with typical angina developed an MI compared with 44% of men.
- In the Coronary Artery Surgery Study (CASS), only 50% of women with typical angina had significant CAD compared with 83% of men.
- Grauer noted that data from the Framingham Heart Study revealed that for women, the initial manifestation of CAD is usually stable angina (47% in women compared to 26% in men).
- Women having an MI are more likely to present with atypical chest pain (midback pain) and atypical symptoms (indigestion, nausea, vomiting and dyspnea).
    - In this study 88 out of (88%) 100 patients presented with chest pain. Other symptoms
        - Nausea – 81%
        - Vomiting – 61%
    - In a study by **MARRUGAT et al., (1998)** showed that women are more likely to have symptoms such as nausea.
- Among Indian women, the presence of hypertension, diabetes, low levels of high density lipoprotein (HDL) and high levels of total cholesterol (TC), triglycerides (TG), low density lipoprotein (LDL), and Lp(a) are correlated with CAD
- At a given level of risk factors, compared to Americans, the CAD risk is 50% lower among southern Europeans but 50% higher among northern Europeans.

- The risk of CAD among Indians is even greater than in northern Europeans at any given level and/or combination of conventional risk factors
    - In this study 45% presented with elevated Fasting Blood sugar Level.
    - In this study 50% presented with elevated Postprandial glucose level.
    - 37% found to have elevated cholesterol levels
    - 57% found to have decreased HDL levels
    - 59% found to have high triglycerides levels
    - 40% found to have high LDL levels
    - 25% patients presented with High blood pressure.

- ✓ Hypertension confers a 4-fold risk of CAD in women versus a 3-fold one in men
- ✓ In the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III), diabetes is regarded as a CAD risk equivalent.
- ✓ Diabetes confers substantial increase relative risk of first, incident, and admission for MI for women with age-to-sex matched controls younger than age 65 yrs over 20 yrs follow up in **COPENHAGEN CITY HEART STUDY**.
- ✓ The INTERHEART study reported that the ratio of apolipoprotien (apo B to apo A-1) was an important risk marker for acute myocardial infarction. Higher levels were seen in South Asian cases.

- ✓ The **JAIPUR HEART WATCH** (JHW) studies reported that there is a significant increase in total cholesterol, LDL cholesterol and triglycerides and a decline in HDL cholesterol is women and men at all age groups.

- ✓ **GUPTA et al** reported in Jaipur hypertension in females are high and **JOSEPH et al** reported in Trivandrum reported SHT in females are high compared to male and increase the risk of CAD.

- ✓ **HUXLEY AND COLLEAGUES** found women with diabetes has 3.5 fold increase in cardiovascular mortality compared with non-diabetic women as well as their male counterpart.

- ✓ Cardiovascular disease is the most common complication of diabetes in women. Women with DM have CAD incidence similar to men, irrespective of age.

- ✓ The risk of CAD among diabetic subjects is remarkably higher compared to non diabetic subjects. The risk of death due to CAD in diabetic subjects with one prior myocardial infarction is similar to that sex in a non diabetic subjects with an earlier MI, while the risk is tri-placed in diabetic subjects with known MI. The life expectancy of diabetic patient is reduced by 30% compared to non diabetic subjects which translates to 8 year of loss of life in diabetic subjects. Further the protection in pre-menopausal women is abolished in diabetic premenstrual women.

- ➢ Physically active women have a 50% lower risk of CAD than sedentary women.

- ➢ The annual prevalence of obesity among U.S. adults age 20 and older has increased from 19.4% in 1997 to 23.9% in 2002 (Centers for Disease Control and Prevention, 2003). For

the first six months of 2003, the prevalence of obesity among women was highest among non-Hispanic black women (38.7%), followed by Hispanic or Latino women (25.7%).

- A sedentary lifestyle is the most common risk factor for coronary disease in women, Grauer said, and as a group, women are less active then men. However, 30 to 45 minutes of walking three times a week could reduce the risk of MI by as much as 50%.

- In this study 89% reported sedentary habits. Major risk factors for CAD dependent on the demographic and societal transition are physical inactivity, excess dietary calories and fat intake, being overweight/obese, high blood pressure, diabetes, cholesterol levels, the metabolic syndrome and psychosocial stress.

    - In this study 51% were found to have over weight
    - In this study 23% were found to have obesity
    - In this study 51% presented with >0.85 WHR.
    - Obesity is linked to multiple cardiac risk factors like DM, SHT, Dyslipidemia.

In this study

- In our study prevalence of smoking among women was zero percent.
- A study **LIAQUAT ALI CHEEMA et al**, (Gender comparison of coronary risk factors and clinical presentation in Paskistani patients with coronary artery lesion).
- This showed that smoking was not a risk factor is females in the study population and diabetes mellitus and obesity were the common factors for CAD.

- In a study by **V.CHIAMVIMONSAT** and **L.STERNBERG** (University of Toronto)
- Coronary artery disease is the leading cause of mortality in women, with incidence after menopause equal to that of men.
- Diabetes and post menopausal status are the strongest risk factors.

List of Findings

Age distribution	Between 40-50yrs	17%
	Between 50-60yrs	24%
	Between 60-70yrs	33%
	Between 70yrs	26%
BMI	Healthy	26%
	Overweight	51%
	obese	23%
WHR	>0.85	81%
	<0.85	19%
Menstruation	7 patients were menstruating women	
Sedentary habits	89%	
H/O OCP intake	40%	
Clinical symptoms	Chest pain	88%
	Nausea	81%
	Vomiting	62%
	Syncope	50%
	Radiation of pain	31%
	Palpitation , epigastric pain	29%
Clinical signs	Lung Basal Crepitations	45%
	Elevated JVP	23%
	S3	16%
	S4	24%
	Wheeze	8%
	Hypotension	10%

Type 2DM	Fasting blood sugar	45%
	Post prandial blood sugar	50%
	Newly Detected	20%
	Known Type 2DM	30%
SHT	25%	
Lipid abnormalities	High cholesterol	37%
	Low HDL levels	57%
	High LDL levels	40%
	High triglycerides	59%
CRP	Elevated CRP	71%
ECG abnormalities	ST Elevation MI	80%
	NON ST Elevation MI	10%
	Unstable Angina	10%
Troponin in NSTEMI/UA	Positive in 10 patients	
Thrombolysed	21%	

Risk	**AHA'S National Study**	**In this Study**
Overweight	41%	51%
Sedentary habits	40%	89%
Smoking	36%	Nil
High Cholesterol	31%	37%
SHT	19%	25%
DM	7%	50%
TGL	1%	59%

7. CONCLUSION

The following are the conclusions that could be inferred from this study on clinical spectrum and risk factors for CAD among female patients.

1. The most common presentation is chest pain.
2. The most common cardiovascular sign is Lung Basal Crepitations.
3. WHR associated with obesity and overweight increase the risk of Myocardial Infarction in female population
4. Most of the patients with Myocardial Infarction have dyslipidaemia.
5. Diabetes Mellitus clearly related to Myocardial Infarction during the premenopausal period.
6. Hypertension is also associated with increased risk of Myocardial Infarction.
7. Sedentary habits are associated with increased risk of coronary artery disease.
8. Most common presentation is STEMI.
9. Among STEMI most common type of MI is ASMI.
10. OCP intake accounts for risk of being overweight and causes MI.

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					CLINICAL FEATURES																													
					SYMPTOMS																															
			HT	WT	BMI	CP	N	VO	S	R	DY	O	JVP	S3	S4	CREPT	WHZ	SHT	DM		LIPID				SMO	ALC	MENS	SED	ECG	ECHO	TRO	THR	WHR	OCP	CRP
S.NO																			F	PP	TC	TG	LDL	HDL											
1	40	24991	142	63	31.2	Y	Y	N	N	N	N	Y	Y	N	N	Y	N	130/90	140	196	222	300	170	168	N	N	N	Y	Y	N	ND	Y	Y	Y	Y
2	45	24951	139	49	28.4	Y	Y	Y	N	Y	N	N	N	N	N	N	N	160/98	90	130	160	130	112	38	N	N	Y	N	Y	N	ND	Y	Y	Y	N
3	43	24812	141	70	35.2	Y	Y	N	N	N	N	N	N	N	N	N	N	120/70	170	220	202	186	168	37	N	N	Y	N	Y	N	ND	Y	Y	Y	N
4	43	26244	153	69	29.5	Y	N	N	N	Y	N	Y	Y	Y	N	Y	N	150/90	80	180	142	130	112	32	N	N	Y	Y	Y	Y	ND	Y	Y	Y	N
5	69	26360	137	46	24.5	Y	Y	N	N	N	N	N	N	N	N	N	N	160/90	76	120	140	130	107	33	N	N	N	N	Y	Y	ND	Y	Y	N	N
6	65	27745	152	45	19.5	Y	Y	N	N	Y	N	N	N	N	N	N	N	120/70	250	160	200	140	98	30	N	N	N	Y	Y	Y	ND	Y	N	N	N
7	63	27777	138	66	34.7	Y	Y	N	N	Y	N	N	N	N	N	N	N	130/80	180	330	220	340	170	64	N	N	N	Y	Y	Y	ND	Y	Y	N	Y
8	65	31327	142	57	28.3	Y	Y	Y	Y	Y	N	N	N	N	N	N	N	140/80	150	140	180	120	98	32	N	N	N	Y	Y	Y	ND	Y	Y	N	N
9	56	31643	141	70	35.2	Y	Y	N	N	Y	N	Y	N	N	N	N	N	190/90	90	126	140	180	108	34	N	N	N	Y	Y	Y	ND	Y	Y	N	N
10	65	32160	142	68	33.8	N	Y	N	Y	N	N	N	N	N	N	N	N	170/98	90	120	200	176	118	36	N	N	N	Y	Y	Y	ND	Y	Y	N	N
11	60	32781	140	53	27	N	Y	N	Y	N	N	Y	N	N	N	Y	N	190/100	86	132	198	330	162	68	N	N	N	Y	Y	Y	ND	Y	Y	N	Y
12	55	34192	144	59	25.5	Y	Y	Y	Y	N	N	N	N	N	N	N	N	130/80	76	118	198	300	167	67	N	N	N	Y	Y	Y	ND	Y	Y	N	Y
13	55	34229	148	54	24.7	Y	Y	Y	N	Y	N	N	N	N	N	N	N	120/80	90	112	248	330	161	66	N	N	N	Y	Y	Y	ND	Y	Y	N	Y
14	70	35771	143	79	38.6	Y	Y	Y	Y	Y	N	N	N	N	N	N	N	110/70	80	122	270	350	180	64	N	N	N	Y	Y	Y	ND	Y	Y	N	Y
15	70	36237	147	55	25.5	Y	Y	Y	N	N	N	N	N	N	N	N	N	110/70	76	132	208	300	163	67	N	N	N	Y	Y	Y	ND	N	Y	N	Y
16	55	34181	150	63	28	Y	Y	Y	Y	Y	N	Y	Y	Y	N	Y	N	90/70	79	129	200	202	162	68	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
17	80	38457	141	54	27.1	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	80/60	90	330	200	408	165	72	N	N	N	Y	Y	Y	ND	N	Y	N	Y
18	80	38512	151	47	20.6	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	90/70	190	330	190	112	122	36	N	N	N	Y	Y	Y	ND	N	N	N	N
19	75	38584	143	54	26.4	Y	N	N	N	N	N	N	N	N	N	N	N	110/70	180	340	180	100	126	38	N	N	N	Y	Y	Y	ND	N	Y	N	N
20	75	397773	146	62	29.1	Y	N	N	N	N	N	N	N	N	N	N	N	120/80	110	140	208	300	171	66	N	N	N	Y	Y	Y	ND	N	Y	N	Y
21	62	40070	155	56	23.3	Y	Y	Y	N	N	N	N	N	N	N	N	N	130/80	96	126	220	316	182	69	N	N	N	Y	Y	Y	ND	N	N	N	Y
22	70	40310	141	66	33.2	Y	Y	Y	N	N	N	N	N	N	N	N	N	120/80	110	130	280	314	201	64	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
23	85	44657	139	58	30	Y	N	N	N	Y	N	N	N	N	N	N	N	120/80	80	126	180	300	207	66	N	N	N	Y	Y	Y	ND	N	Y	N	Y
24	60	45053	152	54	23.4	Y	Y	Y	Y	Y	Y	N	N	N	N	N	N	130/80	90	130	174	400	162	64	N	N	N	Y	Y	Y	ND	N	Y	N	Y
25	74	44863	143	70	34.2	Y	N	Y	Y	Y	N	N	N	N	N	N	N	120/80	78	138	200	110	163	38	N	N	N	Y	Y	Y	ND	N	Y	Y	N
26	40	45095	151	53	23.2	Y	Y	Y	N	N	N	N	N	N	N	N	N	110/80	88	128	210	110	126	38	N	N	N	Y	Y	Y	ND	N	Y	N	N
27	80	46086	140	56	28.6	Y	Y	Y	Y	N	N	N	N	N	N	N	N	120/80	186	330	110	304	161	64	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
28	75	45206	144	55	26.5	Y	Y	N	N	Y	N	N	N	N	N	N	N	144/90	190	320	188	318/	182	62	N	N	N	Y	Y	Y	ND	N	Y	N	Y
29	82	46041	140	67	34.1	Y	Y	Y	Y	N	N	Y	N	Y	N	Y	N	170/80	180	330	200	380	196	14	N	N	N	Y	Y	Y	ND	N	Y	N	Y
30	82	47364	153	71	30.3	Y	Y	Y	S	N	Y	Y	Y	Y	Y	Y	N	200/100	84	128	208	110	125	36	N	N	N	Y	Y	Y	ND	N	Y	N	N
31	50	48760	148	58	26.5	N	Y	N	N	N	N	N	N	N	N	N	Y	110/80	74	128	210	333	196	66	N	N	N	Y	Y	Y	ND	N	Y	N	Y
32	65	48719	145	55	26.2	N	Y	N	Y	N	N	N	N	N	N	N	N	130/80	70	130	280	400	190	69	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
33	70	48852	153	65	27.8	Y	Y	Y	Y	Y	Y	Y	N	N	N	Y	N	90/70	90	130	200	408	182	69	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
34	55	49212	147	58	26.8	Y	Y	Y	Y	Y	Y	N	N	N	N	N	N	150/90	78	126	114	340	172	67	N	N	N	Y	Y	Y	ND	N	Y	N	Y
35	85	1760	152	61	29.4	Y	Y	Y	N	Y	Y	Y	N	N	N	Y	N	160/90	80	120	200	300	193	63	N	N	N	Y	Y	Y	ND	N	Y	N	Y
36	72	2968	162	50	19	Y	Y	N	N	Y	N	N	N	N	N	N	N	90/70	190	300	202	108	122	34	N	N	N	N	Y	Y	ND	N	Y	N	N
37	65	2970	157	60	26.3	N	Y	N	Y	N	N	N	N	N	N	N	N	120/90	148	294	198	300	112	63	N	N	N	N	Y	Y	ND	N	Y	N	Y
38	68	4415	142	49	24.3	Y	Y	Y	Y	N	N	N	N	N	N	N	N	160/80	136	196	228	312	114	63	N	N	N	N	Y	Y	ND	N	N	N	Y
39	65	4493	142	50	22.5	Y	Y	Y	Y	Y	N	N	N	N	N	N	N	110/90	148	222	180	302	128	65	N	N	N	N	Y	Y	ND	N	Y	Y	Y
40	48	5805	152	54	23.3	Y	Y	Y	Y	N	N	N	N	N	N	N	N	130/80	200	404	172	376	193	67	N	N	N	N	Y	Y	ND	N	N	Y	Y
41	50	5977	154	51	21.5	Y	Y	Y	Y	Y	N	N	N	N	N	Y	N	120/80	110	130	148	140	116	34	N	N	N	N	Y	Y	ND	N	N	Y	N
42	55	7064	148	58	26.4	Y	N	Y	Y	N	N	N	N	N	N	Y	N	120/80	96	118	222	132	114	34	N	N	N	N	Y	Y	ND	N	Y	Y	N
43	79	7125	150	65	28.8	Y	Y	Y	N	Y	Y	N	N	N	N	Y	N	130/100	100	122	202	118	112	34	N	N	N	N	Y	Y	ND	N	Y	N	N

					CLINICAL FEATURES																												
					SYMPTOMS																												
			HT	WT	BMI	CP	N	VO	S	R	DY	O	JVP	S3	S4	CREPT	WHZ	SHT	DM		LIPID				SMO	ALC	MENS	SED	ECG	ECHO	TRO	THR	WHR	OCP	CRP
																			F	PP	TC	TG	LDL	HDL										
44	42	8454	149	63	28	N	N	Y	Y	Y	Y	N	N	N	N	Y	N	130/80	186	336	170	300	142	66	N	N	Y	Y	Y	Y	ND	N	Y	Y	Y
45	65	8589	145	55	26	Y	Y	Y	Y	Y	Y	N	N	N	N	Y	N	160/80	172	316	156	312	147	66	N	N	Y	Y	Y	Y	ND	N	Y	N	Y
46	66	8606	153	65	28	Y	Y	Y	N	Y	N	N	N	N	N	Y	N	130/80	90	110	156	302	152	64	N	N	Y	Y	Y	Y	ND	N	Y	N	Y
47	65	11245	150	63	28	Y	Y	Y	Y	N	N	Y	Y	Y	N	N	N	180/100	78	128	148	376	196	64	N	N	Y	Y	Y	Y	ND	N	Y	N	Y
48	46	11709	147	58	27	Y	Y	Y	N	N	N	Y	N	Y	N	N	N	130/90	90	110	202	140	102	34	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
49	67	11811	153	67	29	Y	N	N	N	N	N	Y	N	N	N	N	N	190/100	70	110	220	132	108	34	N	N	N	Y	Y	Y	ND	N	Y	N	Y
50	63	12668	144	48	23	Y	N	N	N	Y	N	N	Y	N	N	Y	Y	130/80	202	336	200	118	106	38	N	N	N	Y	Y	Y	ND	N	Y	N	Y
51	70	15613	149	56	25	Y	N	N	N	Y	N	Y	N	Y	N	Y	N	160/90	176	286	244	306	98	39	N	N	N	Y	Y	Y	ND	N	N	N	Y
52	42	15797	150	61	27	Y	N	N	Y	N	Y	N	Y	N	N	Y	N	200/100	174	296	202	318	94	37	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
53	85	15856	152	68	29	N	Y	Y	N	N	N	Y	N	N	N	Y	N	200/100	166	316	220	122	112	35	N	N	N	Y	Y	Y	ND	N	Y	N	Y
54	48	17168	149	49	22	N	N	Y	N	Y	N	N	Y	N	N	N	N	120/80	174	348	200	116	116	35	N	N	N	Y	Y	Y	ND	N	N	Y	Y
55	65	16830	147	52	29	Y	N	Y	Y	N	Y	N	N	N	Y	N	N	190/100	114	388	244	116	117	35	N	N	N	Y	Y	Y	ND	N	Y	N	Y
56	80	17314	146	59	28	Y	Y	Y	Y	N	N	N	N	N	N	N	N	120/80	110	306	202	108	104	34	N	N	N	Y	Y	Y	ND	N	Y	N	N
57	79	17809	162	50	19	Y	Y	Y	N	Y	N	N	N	N	N	N	N	160/80	90	228	198	208	104	36	N	N	N	Y	Y	Y	ND	N	N	N	N
58	65	14301	143	47	23	Y	Y	Y	N	Y	Y	N	N	N	N	Y	N	130/80	76	110	148	300	106	30	N	N	N	Y	Y	Y	ND	N	N	N	N
59	91	18951	158	59	24	N	Y	Y	N	N	Y	Y	N	Y	N	Y	N	120/80	202	126	178	316	111	28	N	N	N	Y	Y	Y	ND	N	N	N	N
60	55	19020	153	66	28	Y	N	N	Y	N	Y	Y	N	Y	Y	Y	N	110/80	90	336	178	380	144	66	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
61	72	19032	147	63	28	Y	Y	Y	N	N	Y	N	Y	Y	Y	Y	N	110/70	222	110	179	378	144	68	N	N	N	Y	Y	Y	ND	N	Y	N	Y
62	55	21187	151	60	26	Y	Y	N	Y	Y	N	N	Y	Y	Y	Y	Y	90/60	200	401	190	306	117	69	N	N	Y	Y	Y	Y	ND	N	N	Y	Y
63	60	22478	147	61	28	Y	Y	Y	Y	N	N	Y	N	N	Y	Y	N	90/60	174	290	190	308	121	72	N	N	Y	Y	Y	Y	ND	N	N	Y	Y
64	58	22486	153	58	25	Y	Y	Y	N	Y	N	Y	N	N	N	Y	Y	80/80	180	298	190	310	126	28	N	N	Y	Y	Y	Y	ND	N	Y	Y	Y
65	72	24777	149	62	28	Y	Y	N	N	Y	N	Y	Y	N	N	N	N	110/70	78	398	222	130	126	32	N	N	Y	Y	Y	Y	ND	N	Y	N	Y
66	70	75290	145	69	33	Y	Y	N	N	N	N	Y	Y	N	N	N	N	110/70	70	128	240	130	128	32	N	N	N	Y	Y	Y	ND	N	Y	N	Y
67	72	75648	152	55	24	Y	Y	N	N	N	N	Y	Y	N	Y	N	N	120/80	90	172	200	100	126	30	N	N	N	Y	Y	Y	ND	N	N	N	Y
68	70	662	153	70	30	Y	Y	N	Y	N	Y	Y	Y	N	Y	Y	N	110/70	78	128	202	300	126	28	N	N	N	Y	Y	Y	ND	N	Y	N	Y
69	60	1036	149	57	26	Y	Y	Y	Y	N	N	Y	Y	N	Y	Y	N	90/60	80	132	180	117	129	26	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
70	59	3009	149	57	23	Y	Y	Y	N	N	N	Y	Y	N	Y	Y	N	100/70	90	140	188	330	155	65	N	N	N	Y	Y	Y	ND	N	N	Y	Y
71	45	3214	147	63	29	Y	Y	Y	N	N	N	N	Y	N	Y	Y	N	130/80	186	333	176	340	145	65	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
72	60	3258	150	70	31	Y	Y	N	N	N	N	N	N	N	N	Y	N	110/70	176	380	176	370	147	67	N	N	Y	Y	Y	Y	ND	N	Y	Y	Y
73	60	3282	148	54	25	Y	Y	N	N	N	N	N	N	N	N	N	N	120/80	200	342	190	374	212	69	N	N	Y	Y	Y	Y	ND	N	N	N	Y
74	75	4599	151	64	29	Y	Y	N	N	N	N	N	N	N	Y	N	N	120/80	192	400	190	110	118	26	N	N	Y	Y	Y	Y	ND	N	Y	N	N
75	75	4679	150	63	28	Y	Y	N	N	N	N	N	N	N	Y	Y	N	110/80	92	120	176	110	116	28	N	N	Y	Y	Y	Y	ND	N	Y	N	Y
76	85	3955	147	58	27	Y	Y	N	N	N	Y	N	N	N	Y	Y	N	130/80	112	140	220	116	104	32	N	N	Y	Y	Y	Y	ND	N	Y	Y	Y
77	50	4707	151	62	27	Y	Y	N	N	N	Y	N	N	N	Y	Y	N	200/100	84	150	220	116	102	32	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
78	45	4593	154	51	22	Y	Y	N	N	N	Y	N	N	N	Y	Y	N	110/80	120	190	300	122	98	32	N	N	N	Y	Y	Y	ND	N	N	Y	Y
79	70	4799	147	63	29	Y	Y	N	N	N	N	N	N	N	Y	Y	N	120/80	122	160	245	110	96	34	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
80	60	6013	151	60	26	Y	Y	Y	Y	Y	Y	N	N	N	N	Y	N	130/80	95	130	165	110	90	33	N	N	N	Y	Y	Y	ND	N	Y	Y	Y
81	57	7282	144	59	29	Y	Y	Y	Y	N	N	N	N	N	N	Y	N	170/100	110	180	220	330	147	29	N	N	N	Y	Y	Y	Y	N	Y	Y	Y
82	65	7246	148	54	25	Y	Y	Y	Y	N	N	N	N	N	N	Y	N	150/90	160	224	200	314	149	31	N	N	N	Y	Y	Y	Y	N	Y	Y	Y
83	75	10077	141	70	35	Y	Y	Y	Y	Y	N	N	N	N	N	Y	N	190/100	220	336	222	400	100	72	N	N	N	Y	Y	Y	Y	N	Y	N	Y
84	60	10239	150	63	28	Y	Y	Y	N	N	N	N	N	N	N	N	N	120/80	110	180	180	130	106	29	N	N	N	Y	Y	Y	Y	Y	Y	Y	N
85	45	10142	139	58	80	Y	Y	Y	N	N	N	Y	Y	N	N	N	N	120/80	146	228	186	120	109	29	N	N	Y	Y	Y	Y	Y	Y	Y	Y	N

					CLINICAL FEATURES																													
					SYMPTOMS																															
			HT	WT	BMI	CP	N	VO	S	R	DY	O	JVP	S3	S4	CREPT	WHZ	SHT	DM		LIPID				SMO	ALC	MENS	SED	ECG	ECHO	TRO	THR	WHR	OCP	CRP
																			F	PP	TC	TG	LDL	HDL											
86	70	13073	182	54	24	Y	Y	Y	Y	Y	N	N	N	N	N	N	N	120/80	130	196	146	150	104	31	N	N	N	Y	Y	Y	Y	Y	N	N	Y
87	64	14757	143	70	34	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	130/78	200	332	200	314	155	64	N	N	N	Y	Y	Y	Y	Y	Y	N	Y
88	80	1486	140	56	29	Y	Y	Y	Y	Y	Y	N	Y	N	N	N	N	110/70	108	138	184	129	113	29	N	N	N	Y	Y	Y	Y	Y	Y	N	Y
89	48	18521	144	55	27	Y	N	N	Y	N	N	N	N	N	N	N	N	130/80	107	127	180	107	119	27	N	N	N	Y	Y	Y	Y	Y	Y	Y	Y
90	54	18872	140	67	34	Y	N	N	N	Y	N	N	Y	N	N	N	N	130/78	188	290	222	300	158	62	N	N	N	Y	Y	Y	Y	Y	Y	Y	Y
91	60	22088	141	70	35	Y	Y	Y	N	N	Y	N	N	N	N	N	N	124/70	144	212	177	122	117	34	N	N	N	Y	Y	Y	N	N	Y	Y	Y
92	70	22765	153	71	30	Y	N	N	Y	Y	N	Y	N	Y	Y	N	N	170/100	144	288	198	180	117	34	N	N	N	Y	Y	Y	N	N	Y	N	Y
93	65	12636	140	67	34	N	Y	Y	Y	N	N	N	N	N	N	N	N	110/70	166	300	202	301	104	36	N	N	N	Y	Y	Y	N	N	Y	N	Y
94	54	23229	140	56	29	Y	Y	Y	Y	Y	Y	N	N	N	N	Y	N	100/80	110	140	212	308	156	64	N	N	N	Y	Y	Y	N	N	Y	Y	Y
95	60	27411	151	53	23	N	N	Y	Y	N	N	N	N	N	N	Y	N	110/80	90	110	300	412	106	66	N	N	N	Y	Y	Y	N	N	N	N	N
96	65	2679	152	54	23	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	Y	90/70	218	412	132	228	121	34	N	N	N	Y	Y	Y	N	N	N	N	N
97	75	27811	179	58	30	Y	Y	Y	Y	Y	Y	N	N	N	N	N	N	110/80	110	132	170	140	128	34	N	N	N	Y	Y	Y	N	N	Y	N	N
98	64	28002	147	55	26	Y	Y	Y	Y	Y	Y	N	N	N	N	N	N	120/80	106	126	110	156	121	36	N	N	N	Y	Y	Y	N	N	Y	Y	N
99	55	30942	138	66	35	N	N	Y	Y	N	N	N	N	N	N	N	N	120/80	98	116	94	122	111	38	N	N	N	Y	Y	Y	N	N	Y	Y	N
100	70	26719	141	70	35	Y	Y	Y	Y	Y	Y	Y	N	N	N	N	N	120/80	90	110	90	130	113	36	N	N	N	Y	Y	Y	N	N	Y	N	Y

CP -Chest Pain
N - Nausea
V - Vomiting
S - Syncope
O - Others
Crept - Crepitation
WHZ - Wheeze
SMO - Smoking
ALC - Alcohol
MENS - Menstruation
SED - Sedentary Habits
TRO - Troponin
THR - Thrombolysed
WHR - Waist Hip Ratio
OCP - Oral Contraceptive Pills
ND - Not Done

INFORMED CONSENT

Department of General Medicine

Coimbatore Medical College, Coimbatore

Principal Investigator : **Dr. E.ARUL ANANDHAN**

Research guide : **Dr. S.USHA**

Organization : Department of General Medicine

Informed Consent : I have been invited to participate in research project titled

'CORONARY ARTERY DISEASE IN FEMALES'

I understand it will involve answering a set of questionnaire, undergo physical examination and basic investigations.

Also I give consent to utilize my personal details for study purpose and can be contacted if necessary.

I am aware that I have the right to withdraw at any time which will not affect my medical care.

Name of participant :

Signature :

Date :